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ABSTRACT

A collection of papers on neurolinguistics includes: "Communication Strategies in Aphasia" (Elisabeth Ahlsen); "Speech Planning in the Light of Stuttering" (Ann-Marie Alme); "L. S. Tsvetkova's Aphasia Rehabilitation Method and Its Applications" (Ritva Hanninen); "Semantic Aphasia and Luria's Neurolinguistic Model" (Birgitta Johnsen); "Aphasic Speech Errors and Their Linguistic Interpretation" (Pirkko Kukkonen); "Word Fluency: A Task Analysis" (Matti Laine); "Speech and Language Disorders in a Dialysis Encephalopathy Patient and the Effect of Desferrioxamine and Reverse-Osmosis Water Treatment" (Matti Lehtihalmes, Reijo Aulanko, Martin von Bonsdorff); "Language Impairment in Alzheimer's Disease and Vascular Dementia" (Maire Lempinen, Raimo Sulkava, Timo Erkinjuntti); "Neurolinguistic Aspects of Finnish Posterior Aphasia" (Jussi Niemi, Paivi Koivuselka-Sallinen); "Fragile X Speech in Finnish: Phonological Observations" (Jussi Niemi, Erkki Vilkman, Unto Ikonen); "The Great Neurolinguistics Methodology Debate" (Loraine Obler); "Clinical Logopedics: A Diagnostical Review of Speech and Language Disorders in a Group of High-Risk Children" (Marjatta Parre, Ulla Strom, Katarina Michelsson); and "Aphasia Syndromes and Their Recovery" (Ivar Reinvang). (MSE)

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N:o 42

NEUROLINGUISTIC PAPERS: PROCEEDINGS OF THE 2ND FINNISH CONFERENCE OF NEUROLINGUISTICS, JOENSUU 1985

edited by

Jussi Niemi & Päivi Koivuselkä-Sallinen

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PREFACE

The organizers were fortunate enough to be able to print all but one of the papers presented at the Second Finnish Conference of Neurolinguistics held at the University of Joensuu, May 31 and June 1 1985. Two of the present papers, those of Prof. Loraine Obler and Dr. Ivar Reinvang, were given as Invited Lectures during the meeting. In addition to the papers, the conference program included a workshop on aphasia rehabilitation as well as two video tape demonstrations on the same topic.

We would like to acknowledge the financial support of the Academy of Finland in organizing the meeting as well as the secretarial help received from Ms. Liisa Enroth in typing the papers.

Joensuu, October 1985

JN & PK-S

COMMUNICATION STRATEGIES IN APHASIA

Elisabeth Ahlsén
University of Göteborg

1. WHY STUDY APHASICS IN CONVERSATION? - SOME PROBLEMS AND WAYS OF DEALING WITH THEM

Linguistic studies of aphasia have traditionally been focusing on specific details as parts of the communicative ability, studying these details mainly in a test setting. Therefore we lack information about the behaviour of aphasics in conversation. This, of course, can be dealt with by doing more studies of the same factors which have been studied in tests, but in a conversation setting. We can ask questions like "What is the correlation of the difficulties found in tests and in conversations?", "What additional knowledge can we obtain by conversation studies?" and "How can this knowledge be used?".

There are great problems concerning which methods to use to be able to handle conversation data in the best way for studying aphasia as well as many other phenomena. These problems tend to have a discouraging effect on those who wish to study such data, using "established" methods, since such methods are still very rare. The design and tentative application of new methods involves time-consuming and cumbersome work, which nevertheless will have to be done. New technical means for this kind of studies are available in video- and audio-recording facilities for obtaining data and in computer programs for doing analyses. But technical aids are not enough. There is also a need of an increased knowledge and more comprehensive theories in the field of conversation analysis. One has to consider which factors might influence behaviour in conversations (cf. Allwood 1984) and how this might happen and investigate these factors systematically.

Through studies of aphasics in conversation we will gain more knowledge a) of aphasia, b) of how the brain works and c) of how communication works. Some relevant questions are: "What happens to communication in different situations for aphasics?", "Why do specific patterns of behaviour show up?", "What possibilities and limitations are there in communication-systems in terms of available means of expression?", "How are these means affected by different types of aphasia and by different situations?" etc. The answers to these questions will eventually have to be

related to acceptable explanations in terms of neuropsychological and neurophysiological theories and these two kinds of theories will have to be adjusted to one another. The parts of new information which will be gained by doing conversation studies can be integrated with what is known about communication and brain damage into a better overall picture.

Today there is a bias in research about aphasia, like in many other fields, favouring an atomistic view and giving specific types of knowledge. If the issues mentioned above are dealt with one can also apply a more holistic view to supplement this knowledge.

The practical/therapeutic need for more knowledge about aphasics in conversation is obvious, since the major goal in aphasia rehabilitation is to diagnose and treat the patient's ability to communicate in everyday life activities. It would be an important help in clinical work to be able to use conversation data for prediction of a patient's difficulties, for planning his/her training and giving information to people in the hospital and to the patient's family and others, in a systematic way.

2. ACTIVITY INFLUENCE ON COMMUNICATIVE ABILITIES IN SOME APHASICS

In some studies made in the research project "Aphasia and spoken interaction" (Dept. of Linguistics, Univ. of Göteborg, Sweden) the lack of correlation between test and conversation performances was striking. Ten aphasics were videorecorded in conversations, telling narratives and performing specific tests (a naming test and a test of nonverbal communication) (see Ahlsén 1985).

The correlations of "noticeable word finding problems", concerning nouns, verbs and adjectives in the three different activities were extremely low (0.5 for test-conversation and 0.37 for narration-conversation, using the Spearman rank correlation coefficient). This is not really surprising, since both what the patient is doing and what the "analyzer" is noticing differs quite a lot between the activities. It is, however, important to remember, since results from naming tests are very often used in order to predict if a patient is going to have word finding problems in conversation, and this seems to be very misleading. There is a general tendency for the aphasics in the study to have less noticeable word finding problems in conversations than in tests, but this does not seem to be generalizable to all patients. There are also great differences in the amount of word finding problems in narration between different patients.

In a study of how much nonverbal communication was used (see Ahlsén 1985), it was found that all the aphasics, except two patients with large frontal lobe lesions, used considerably more nonverbal communication in conversations than nonaphasic persons in similar conversations. Two of the patients, who both used extremely great amounts of nonverbal communication in conversations, did, however, have problems performing a simple test, where they were supposed to show conventionalized gestures and facial expressions for "illustrating" words or phrases. The difficulties were caused mainly by apraxia and perseveration in both patients and this "interference" of additional symptoms would, if such a test was used for prediction of "use of nonverbal communication" in general, have caused misleading conclusions also here.

3. SOME CASE STUDY DATA AS EXAMPLES

Conversation and test results, concerning word finding problems and nonverbal communication, for three patients will be discussed. Examples from conversation are given for each patient below.

(T = therapist, P = patient,
// = pause of 3 seconds or more
<> = scope of nonverbal communication (in left column)
<> = description of nonverbal communication (in right column)
[= overlap between patient's speech and therapist's speech
CAPITAL LETTERS = emphatic stress)

Patient A.

VERBAL:	NONVERBAL:
T: when you came home do you remember	
P: [that [eeeh < >	<shakes head, chin in hand>
yes //	
T: do <you live> [in a house then	<hand forwards>
[eeh I <yes>	<nods>
and // yes // <eh> // last year <eh> //	<hand forwards><hand forwards>
<for> // yes <three months>	<hand forwards>
T: <mm>	<nods>
P: for //	
T: three months ago or	
P: <three months ago last year> < > eh	<hand forwards in steps, three fingers forwards><hand forwards>
// < > yes //	<sighs, hand to nose>
T: you are on <last year>	<nods>
P: <yes>	<nods>
T: is it in <the autumn>	<nods>
P: <yes> // <August November November> <NOVEMBER>	<nods><counts on her fingers> <shows fourth finger>
T: <mhm>	<nods>
P: noo <yes>	<waves hand back and forth>

Patient B.

VERBAL:	NONVERBAL:
P: you are < > you are so	<laughs>
T: were you down for months and so	
P: I yes	
T: <yes>	<nods>
P: <yes>	<smiles>
T: you were <yes>	<nods>
P: <yes> boresome	<nods, smiles>
T: <yes>	<nods>
P: < > aah <but it's like that>	<makes smacking sound> <shakes her head>
T: aah	
P: all finished < >	<hangs head down, tongue out>
T: <yes>	<nods>
P: so	
T: sure you are [very tired	
P: [<so that is so>	<nods>
but now <it is not so bad>	<nods>
that's lycky < >	<smacks, turns head to side>
T: noo	
P: yes we hope < >	<sticks out tongue>

Patient C.

VERBAL:		NONVERBAL:
T:	he's been <doing> that for long	<hand up>
P:	<yes> three four years you see now I him < >	<nods> <gets up, moves forwards with large movements of arms and legs>
T:	yes < >	<laughs>
P:	< >	<continues the same movements>
T:	that's him yes < >	<laughs>
P:	so there we have HIM you see <not ME> but HIM he is good	<points to T>
T:	< > <yes you too I think>	<laughs><points to P>
P:	<not ME HIM>	<raises forefinger>
T:	<I can see where he got it from>	<laughs>
P:	<yes yes> that's sure <yes>	<nods><nods>
T:	I wonder if it isn't so	
P:	yes yes yes < >	<strokes his moustache>

Patient A. (with mainly a parietal lesion) has a hesitant way of speaking in conversation, making many pauses with turn keeping gestures. She often stops and rephrases what she has started out to say. This gives the impression of frequent word finding problems and quite effortful conversation. This patient, however, has very few problems in a naming test, although her conversation gives an "anomic" impression. This means that her test results could not predict the number of noticeable word finding problems in conversation.

Patient B. (with a lesion affecting the insula and the arcuate fasciculus) has quite "fluent" speech in conversation. She has some word finding problems, but keeps the conversation going by using verbal as well as nonverbal communication of feelings and interaction regulating communication. The conversation of this patient sometimes gives quite a "normal" impression. In a naming test, however, she is totally unable to find any of the specific target words. In this case, the naming test could give the impression that it would be hard or impossible for the patient to take part in a conversation. The nonverbal communication test also gives the impression of great difficulties for this patient, since she shows apraxia as well as perseveration in the test activity, whereas she uses a great deal of adequate nonverbal communication in conversation.

Patient C. (with a temporal-parietal lesion) shows a third kind of communicative pattern in conversation, relying heavily on the use of illustrating nonverbal communication. This "strategy" is quite successful in conversation and spontaneous

narration, but cannot be used successfully in more specified test activities. In the naming test nonverbal communication is not accepted. The patient finds it very difficult to produce isolated gestures or facial expressions in the nonverbal communication test, although he can perform pantomime sequences including the same expressions and uses them often in spontaneous conversation.

These three patients all show clear discrepancies in their communicative strategies and in the success of these strategies between different kinds of activities, i.e. test and conversation. This shows that the type of activity has to be considered, when one tries to estimate the degree and type of aphasic symptoms in a patient. The type of aphasia and the demands of the activity seem to interact in determining the communicative strategies or patterns shown by different aphasics.

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Ahlsén, E. 1985. Discourse patterns in aphasia, Gothenburg Monographs in Linguistics 5, Department of Linguistics, University of Göteborg.

Allwood, J. 1984. On relevance in spoken interaction. In G. Kjellmer & S. Bäckman (eds.), Papers on Language and Literature, Gothenburg Studies in English 60, Department of English, University of Göteborg.

SPEECH PLANNING IN THE LIGHT OF STUTTERING

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1. INTRODUCTION

The phenomenon of stuttering has confounded clinicians and scientists for literally hundreds of years. It still remains a clinical problem and a scientific challenge to those who are interested in deviant speech processes.

Speech is an important psychological activity, where we in interaction with other people are able to express feelings, thoughts and attitudes etc. and to give specific information. A communicative handicap such as stuttering does not only influence the speech itself, but also speech planning and the stutterer's interaction with other individuals. A person who stutters breaks the normal rules for interaction processes and there is an imbalance in the communication.

It is a well known fact that stuttering is highly dependent on contextual factors. Not only is stuttering behavior different from person to person, it even differs within the same person from moment to moment. Linguistic factors seem to be involved in stuttering, as well as in normal disfluency (Kaasin & Bjerkan, 1982; Van Riper, 1982). One linguistic characteristic, the rhythm of speech, has been the focus of several studies. Wingate (1976, p. 260) viewed stuttering as a "prosodic defect", a defect in the transition to stressed syllables. He also pointed out the lack of research in other languages than English (Wingate, 1979).

The purpose of the present study was to investigate overt audible speech behavior in a group of nine stutterers under different conditions of experimental manipulation. The questions raised considered the relationship between:

- disfluency and speaking modality
- type of disfluency in mild vs. severe stutterers
- disfluency and psycholinguistic variables

In order to approach the above issues it is important to provide an operational definition of stuttering in speech. The various types of disfluency are classified into

nine categories. The classification system is partly based on material presented by Johnson (1961) and Wingate (1964).

PWR - Part-Word Repetition: one or more repetitions of an element equal to or less than a syllable (including monosyllabic words).

AP - Audible Prolongation: audible prolongation of a phonetic segment.

SP - Silent Prolongation: tensely articulated stop closures (blocks). (In the larynx or somewhere else in the vocal tract.)

WWR - Whole-Word Repetitions: one or more repetitions of a word (not including monosyllabic words).

PR - Phrase Repetition: repeated utterance of two or more words.

I - Interjection: insertion of an extra sound before or within a word.

R - Revision: modification of phrase content.

BW - Broken Word: a word not completely pronounced.

HP - Hesitation Pause: an unusual un-filled pause which could not be classified as a silent prolongation.

The first three categories (PWR, AP, SP) were chosen as the definition of stutterings. "Stuttering interruptions to the forward flow of speech are primarily intra-morphemic; normal disfluencies are primarily supra-morphemic" (Van Riper, 1982). "...two "kernel" characteristics of stuttering speech can be discriminated: (a) repetitions (audible or silent) of single unit speech elements; and (b) prolongations (audible or silent) "(Wingate 1964, p. 487). Accordingly, the six remaining categories were considered as normal disfluencies.

These definitions can be applied to five psycholinguistic variables:

(1) grammatical class
(2) word length
(3) position within a word
(4) word position in a phrase
(5) syllable stress

2. METHOD

2.1. Subjects

One female and eight male Swedish speaking stutterers ranging in age from 23 to 46 served as subjects. The severity of stuttering varied from mild to severe. None of the subjects was currently involved in any kind of speech therapy.

2.2. Speech Material

In order to vary the speaking modality and cognitive load, the subjects were recorded in three different situations.

Condition I: oral reading task, where the reading material was a 160 word passage from a neutral text about gardening.
Condition II: reading a dialogue of 200 words. The experimenter was in the same room and read part of the dialogue, but the subjects had the longer lines. The text in this dialogue reflected a communicative situation of every day life.
Condition III: this condition should reflect spontaneous speech to a greater extent than the previous one. Subjects had to read a short story silently and then retell it in their own words (about 100 words).

By varying the complexity of the speech material and by progressively introducing more communicative demands and emotional stress an increase in disfluency should result. The phenomenon of adaptation, that is, the tendency to become more fluent in successive readings of the same or similar passages, should thus be avoided.

2.3. Procedure

Subjects were tested individually in a sound proof booth, and they spoke into a freestanding microphone (Sennheiser Mo 211 U). Recordings were made on a Teac R-340 tape recorder outside the booth. The experimenter had visual contact with the subjects.

Previous studies of stuttering (e.g. Young, 1961) have demonstrated that short segments of speech are sufficient to determine severity of stuttering and that

randomly selected samples could be used. Subsequently, from the total material, five phrases containing 4 - 9 words were randomly selected from each condition for analysis. The phrases from condition I and II were the same for each subject, but in the third condition they differed to some extent due to the spontaneous nature of that condition. In order to have an exact delimitation of a phrase and the same sound level for all phrases, a special computer program was used (written by R. Carlsson at KTH, Stockholm). The final test tape consisted of 135 phrases.

The number of disfluencies and stutterings was counted from the audio tape. The syllable was chosen as the reference unit, in preference to the word, since it is relatively invariant in length, thereby giving a more stable basis for comparison. Frequency counts of disfluency and stuttering are expressed in percentages in relation to the total number of syllables (% SyD and % SyS). Each disfluency event was only counted and listed once in the above mentioned classification system. That means, that if a subject had both a prolongation and a repetition in a word, only the most prominent one was counted and put into a category.

3. RESULTS AND DISCUSSION

3.1. Speaking Modality and Disfluency

As a group, the subjects showed an increase in disfluency across conditions. The mean frequency of % SyD was 12 % in the oral reading task, 14 % in the dialogue condition, and 20 % in the spontaneous speech condition. For the same conditions, the mean frequency of % SyS was 11 %, 13 %, and 12 % respectively.

Looking at individual data, however, a somewhat different pattern was indicated. As Figures 1a and 1b clearly show, there was a noticeable difference in disfluency and stuttering between conditions and between subjects. The nine subjects could be said to form two subgroups depending on their disfluency scores. Five of the subjects fell within the range of moderate - severe, with a mean of 21.3 % SyD (MS-group). Four subjects with mild stuttering (7,5 % SyD) formed the M-group.

Of particular interest are the data from the MS-group. This group showed a significant increase in disfluency and stuttering from oral reading condition to dialogue condition ($p < .05$, two-tailed t-test). In condition III with spontaneous speech they remained highly disfluent but the decrease in stuttering is remarkable.

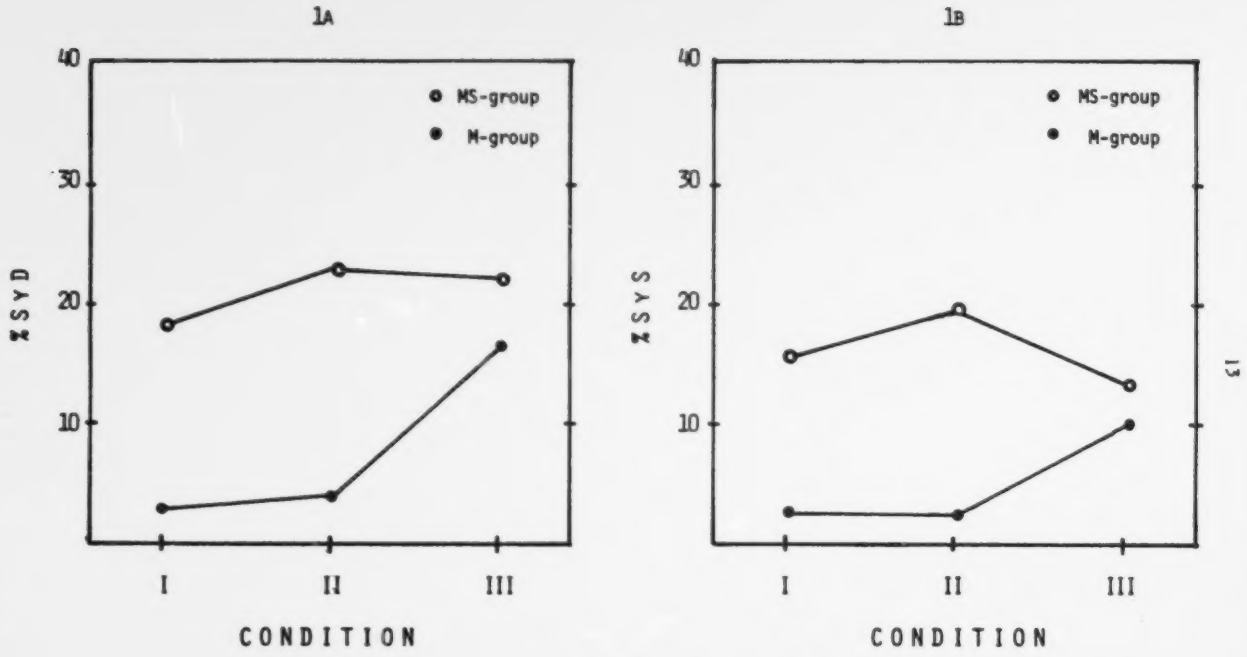

Figure 1A and 1B. Mean percentage of disfluency (% SyD) and stuttering (% SyS) for mild stutterers (M) and moderate-severe stutterers (MS) during three successive speaking tasks; I = oral reading, II = dialogue, and III = spontaneous speech.

The M-group, however, was rather fluent during the first two conditions, but had a significant increase in both disfluency and stuttering in the third condition ($p < .001$).

The between group difference was significant in condition I and in condition II ($p < .001$). The mean frequency of % SyD and % SyS was higher for moderate - severe stutterers even in the third condition compared to mild stutterers, but the difference was not significant.

The dissimilar group trends in fluency break-downs across speech conditions are interesting but difficult to interpret. The dialogue condition was obviously the most "difficult" one for subjects with moderate - severe stuttering. This communicative situation required quick initiation of responses and this fact might have had an increasing effect on expectancy of fear and speech anxiety. Furthermore, the experimenter was sitting in the same room in order to read the opposite lines in the dialogue. This created a more social speaking situation.

Another possible reason for increased fluency failures may be the prosodic factor. Stress and intonation patterns require co-ordination and exact timing and increased activity of the speech mechanism. In Swedish there are two tonal accents, accent I and accent II. In accent II words, the presence of a main stress implies a secondary stressed syllable within the same word. The dialogue task contained more bi- and polysyllabic accent II-words.

The reduced frequency of stuttering for the MS-group in the third condition was remarkable. During spontaneous speech it was possible to avoid certain sounds and "difficult" words by circumlocution and also by using synonyms and fewer polysyllabic words.

3.2. Type of Disfluency

Table I shows the types of disfluencies for all subjects across all conditions. For the total group 78 % of the disfluencies were classified as stutterings. Part-word repetition (PWR) was found to be the most common speech disruption. Subjects with severe stuttering exhibited substantially more repetitions of phonetic segments. They also exhibited instances of stuttering within a word, most of them occuring on stressed syllables. Silent and audible prolongations of sounds (SP and AP) were the second most common type of disfluency. These types were rarely observed in the speech of mild stutterers. 11 % of observed disfluencies were interjections (I). Although insertions of extra sounds or syllables are not uncommon in normal speech, such disruptions are probably sometimes part of the stuttering behavior as they

occur as "starters". Starters can be used before a difficult sound or word to facilitate the utterance, to get started. Gradually the starters will be stereotyped and of no aid anymore. One subject had insertion of "uh"-sounds before and within several words, but with the stringent operational definition of stuttering in this study they were classified as normal disfluencies.

Table I. Distribution of type of disfluency for mild (M) and moderate-severe (MS) stutterers.

	M-group	MS-group	total	%
PWR	27	52	79	33.9
AP	2	48	50	21.5
SP	5	47	52	22.3
WWR	3	1	4	1,7
PR	2	2	4	1.7
I	5	20	25	10.7
R	2	2	4	1.7
BW	2	2	4	1.7
HP	4	7	11	4.7
total	52	181	233	

3.3. Psycholinguistic Variables

3.3.1. Grammatical class. Most of the stutterings occurred on content words (68 %). This finding was most striking for the severe stutterers. Mild stutterers seemed to have a more even distribution of disfluencies between content words and function words. Some studies have indicated that content words are stuttered upon more frequently because they are more meaningful and important, because they have a higher information value and have a tendency to start with a consonant.

3.3.2. Word length. The three word lengths tested were monosyllabic, bisyllabic, and polysyllabic. The mean percentage of fluency failure was analyzed with respect to the occurrence of the words. Looking at the whole group, the results indicated that as syllabic length increases there is a corresponding increase in fluency failures. But, again, there is a difference in the pattern for the subgroups. The MS-

group exhibited a significantly higher rate of disfluency on polysyllabic words than did the M-group.

3.3.3. Position within a word. The following positions were observed: stutterings on the initial sound, the first syllable, and the medial syllable/s. The initial sound and the first syllable are without doubt the most conspicuous positions for fluency failures. The severe stutterers demonstrated far more stutterings on initial sounds, both repetitions and prolongations. They also had several fluency failures within a word, which was never demonstrated by the mild stutterers. The most common for this group was repetition of the first syllable, particularly repetition of monosyllabic words.

3.3.4. Word position in the phrase. The first word, or words early in the sentence, are said to be stuttered upon more frequently than are the remaining words (e.g. Brown, 1938). Clauses analyzed in the present study varied in length from 2 - 9 words (average 5.4 words). In this material, the first three words have only 50 % of the total stuttering occurrences. This is contrary to a majority of other investigations. However, there are some other studies which have shown that the medial part of a sentence had a higher rate of fluency failures and that they sometimes tended to occur in "vollies" on odd numbered words (e.g. Rieber et al, 1976; Soderberg, 1967). This suggests the need for further studies regarding the parameters of stress and intonation patterns in relation to word position.

3.3.5. Syllable stress. A significant difference can be noted between stressed and unstressed syllables. Stutterings occured on 20 % of the stressed syllables compared to only 8 % of the unstressed syllables. It is possible that this partly explains the word position results, since in the speech material for this experiment accented words and stressed syllables did not have an early position in the sentence in most cases. Thus, the fluency failures were most frequent in medial position which tended to be stressed. In Figure 2 the stuttering events are plotted in relation to the total number of stressed and unstressed syllables in all three conditions. An increase in stuttering frequency corresponded to an increase in the difference between syllables. The number of stutterings on stressed syllables was considerable for the MS-group.

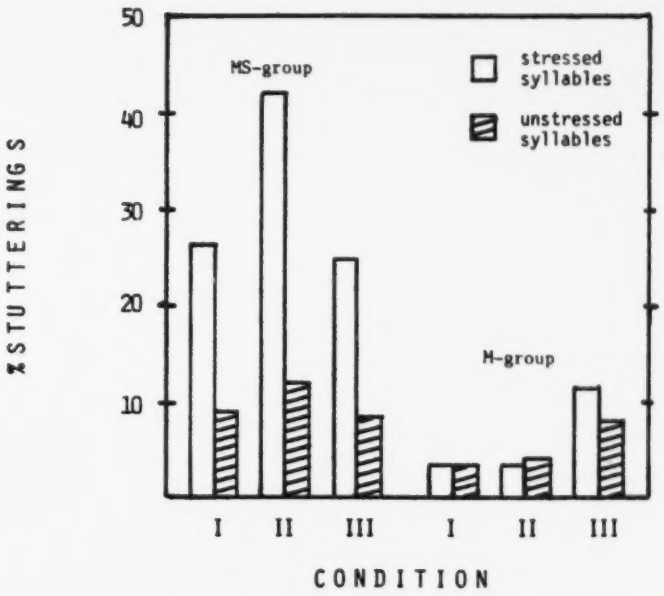

Figure 2. Mean percentage of stuttering in relation to the number of stressed vs. unstressed syllables in oral reading (I), dialogue (II), and spontaneous speech (III); MS-group = moderate-severe stutterers and M-group = mild stutterers.

This sentence is a good example of the observation that fluency failures are not randomly distributed. The crosses mark stuttering events from several subjects.

gäller	inte	samma	regler	på	alla	bussar	då
x	x	x	x	x	x		
x	x		x	x			

The results of the current study thus indicate that varying both speech material and speech tasks was an effective method for elucidation of various speech characteristics in audible speech behavior; both quantity and quality in speech

disruptions were highlighted. In addition, the difference between mild and severe stutterers was demonstrated.

A task of great significance in speech and language research is to investigate characteristics in disordered speech and language. Studies which elucidate linguistic and acoustic characteristics of stuttering will not only be of great clinical importance, but also shed some light on speech planning processes and thus contribute to the research on speech as a window to the brain.

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L.S. TSVETKOVA'S INDIVIDUAL APHASIA REHABILITATION METHOD AND ITS APPLICATIONS

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When considering the questions on which brain mechanisms the higher psychic functions depend, and on which basis their disturbances and reorganization take place, we have to pay attention to the special features of their structure.

In the current view, psychic functions are functional systems as regards their material basis. For example, when a child is born, these systems do not yet exist (like the breathing system does), and they will not arise spontaneously but through the communication process and concrete activity.

A functional system is a uniform and integrated whole, whose material basis is formed by a selected central-peripheric construction. Its composition is not determined by topographical closeness of structures, since its area can include both near-by and distant areas. The main criterion in integrating different areas can be defined as the end-result that it has produced. Every part of this integrated constellation will carry out its own physiological role and this will produce regular features typical of the whole system. These regular characteristics are typical of the whole, but not of the individual parts. Once they are generated these functional systems function as a whole, although one cannot discern their internal make-up. These different type of anatomic and physiological structures will appear only when the system is disturbed. Every functional system is multireceptoric, i.e., it handles a certain amount of afferent signals arriving from the different parts of the brain. The functioning of the system depends precisely on the quality and the amount of the afferent impulses. Although every afferent signal will bring its own special contribution to the functional system, its role will depend on the whole and therefore it will be different in every sub-system. Afferent signals arrive from the so-called afferent area which is generated during ontogenesis. During this process, there will stand out gradually one restricted primary afference, which will carry out the necessary operation of the system. The rest of the afferences, which do have roles of their own in forming the function, fall in a latent state. They do not, however, disappear but they will form a back-up afference group. When the primary afference is disturbed or when the result differs from the normal, these back-up afferences will begin to function again. This state of affairs will exhibit the

integrated character of the functional system. The theory of primary and back-up afferences belongs to the most important theoretical principles in rehabilitating training.

A necessary precondition for a mental function of high quality is that it will at first take place externalized, that is, when the function appears as a detailed, external operation.

Later on the function will shorten in guidance of an adult and it will change its realization level and eventually it will transform into an inner function, in other words, it will be internalized. Internalization of a function, or a gradual change from the outer to inner mental function, is a process that is generated during the human ontogenesis.

Views of disturbancies of the psychic function will thus become comprehensible and we can make the claims:

1. The mass of the secundary and tertiary zones in the brain is not uniform and disturbance or its different parts will have different consequences.
2. The amount of disturbancies of a function is not in a one-to-one relation to the size of the disturbed zone.
3. The consequence of a damage in a specific cortical zone will be a disturbance of some structural part of the psychic function, when the function is disturbed as a whole, because of this system effect. The quality of the disturbance depends on which factor (or a factor of a complicated function in the psychological structure) is damaged. This, for its part, will depend on the location of the lesion.

It is well known that speech disorders can appear both in connection with lesions in the anterior speech zones and in the temporal and parietal zones. In these cases, speech is every time disturbed on a different basis and the structure of the psychological defect will be different. Every psychic function should thus be seen in relation to the whole cortex, and not as separate units. All components are in a close interaction with each other. Although the means in performing functions can vary, the task will remain and the end-result will be constant. This demonstrates the great flexibility and adjustment capacity of the human nervous system.

Soviet neuropsychologists have created a new starting-point for rehabilitation of functions; the reorganization of the functional system in a conscious rehabilitation process.

According to them, rehabilitative training takes place with the help of a reorganization of intra and inter connections of the analyzers. Its basis is formed by

the multimodality of the functional system. When a disturbance occurs, one afferent component can be compensated by another which is resting in a latent state. Rehabilitation takes place with the help of the latent reserves of the damaged functional system.

The human functional systems are so complicated and differentiated that in a difficult functional disturbance, a new functional system can be created only with the help of specific therapy. Also those elements and afferences of the nervous system which have never earlier participated in carrying out the damaged functions will now take part in it.

Luria's investigations are of a particular interest in this area. He was the first to ask the question of the dynamic localization of the functions and of the functional system and to propose a classification of aphasia, which should take into consideration the current view of speech as a complicated functional system. Luria proved that speech is disturbed in different ways according to which factor in the structure has been damaged. Moreover, he formulated the basic principles for rehabilitation and described methods of restoring speech in different aphasias. (See Luria 1970.)

We have attributed to Bein the merit of creating the rehabilitation method of speech in the treatment of vascular aphasia. According to Bein, the most important features of the rehabilitation theory and practice are (see Bein 1964):

1. Importance of phased therapy.
2. Consideration of the semantic area in speech.
3. Differentiation of therapy methods according to form of aphasia.
4. Work which will take all forms of speech into consideration.

Typical of research work done in other countries has been to emphasize the practical side of rehabilitation. It is also typical of these studies that they pay no attention to the nature of the defect and hence the methods will be applied in all cases regardless of the form of the aphasia.

Soviet psychologists have created an effective method to restore the highest cortical functions when the cause of the disturbance of the functions is a local brain damage. The theoretical starting-point is composed of the arguments of the systematic and dynamic localization of the highest cortical functions in the cerebral cortex. Thus it has become possible to re-establish the function to its former condition through regenerating the systems that are the basis of the human psychic function.

On the basis of the systemic and dynamic localization of the functions the consequence of the various kind of brain damages will often be a disturbance of one and the same function. Because of this, it is necessary to make an exact neuropsychological analysis of the damage and to make clear the primary defect at the background. This is inevitable in order to plan the rehabilitation tasks as differentiated units. Because of this, the re-establishing of tasks, the general therapy plan and its concrete procedures must be different in all these cases.

Rehabilitation is building a new functional system upon the preserved afferences. In therapy, one of the most important principles when re-establishing the function is the use of the undamaged analyzers. As mentioned before many various afferent systems during the ontogenesis participate in the arousal process of every higher psychic function. Hence, the function is at first multimodal. Those afferences that have fallen in the reserve are used to compensate every factor, which has fallen off the common system.

For example, a lesion in Wernicke's zone leads to a disturbance of the acoustic component of sound discrimination. In this case the undamaged factor can be replaced with the preserved visual and motor afference. These had participated in the arousal process of sound discrimination and gave lately room to the leading acoustic afference of this function. The patients learn the methods of sound discrimination appropriate in the new situation. For this purpose they use the oral sound and word scheme in the same way as they use the kinesthetic signals, which are sent by the same organs during articulation. E.g. in sensory aphasia a patient learns sound discrimination according to the following programme:

1. The patient listens to the pronunciation of the whole word.
2. He will look for the oral model of the word uttered by therapist.
3. He will imitate without, however, producing, the initial sound.
4. He will control his own oral model using a mirror.
5. He will look for the corresponding letter and will palpate its form with his fingers.
6. He will pronounce the sound and put the single letter in question under the corresponding picture.

When the patient is going through sound discrimination with the previous programme, the external means will be the function which has the following external structure when preceding from word to sound:

pronounced word	oral model of the first sound	letter	sound

The described practices include all preserved analyzers: visual, kinesthetic and kinetic and, in addition to this, it will make use of semantic level of speech.

Furthermore, in rehabilitation, one does not pay attention to multimodality only, but also to the fact that the damaged functions can have influence at different levels. In the cortical damages all the highest levels will weaken first where the functions operate maximally at the subject's own will. The level at which the function is performed automatically, or is less subject to one's own will, will suppress less. The patients are left with such social experience with speech and intellectual functions that has influence at these lower levels. The utilization of these functions strenghtened in the past is therefore one of the most important principles in therapy. When relying upon these strenghtened forms, one is bound to succeed in replacing the disturbed function temporarily to the lower and easier level. In literature, there exists evidence of this kind of method being known for a long time. E.g. in afferent motor aphasia where the elementary speech functions are better preserved, the rehabilitation begins with arousing the involuntary speech processes. In this case methods will be used that arouse the most automatic speech forms. Arousing external speech will begin with exercises that help to actualize very strong series of speech, such as series of numbers, days of a week, familiar songs and enumerating things with one's fingers. It is important to include preserved parts of emotional speech like names of persons close to the patient. Singsong reading of poems that is coupled with finger tapping in order to stress the rhythmical structure will make the arousal of involuntary speech easier.

It has turned out in practice, that the re-established speech function does not always receive an active form. All patients suffering from aphasia do not use expressive speech actively for purposes of communication with their environment after the therapy period. It is often used only with the therapist, because the therapist's means of stimulation makes the communication easier. Therefore rehabilitation has to ensure that the functions will be returned to active and self-controlled form, not only to the passive store of experience. This problem is intimately connected with the demand to create a function model that has to be adopted to suit his needs. Our analysis of existing aphasia therapy forms has proved that the most widely used method of the therapist is to show an example and act as a model. This way some component in the re-establishment of functions can be reached, but this method does not lead to the reorganization of the inner structure of the function and to an active execution of function that has to be re-established.

In rehabilitation there is usually given a list of special uncoordinated methods that are recommended for a specific form of aphasia or for rehabilitation of a defect of a certain degree. An analysis and the preparation of a rehabilitation method are often replaced by a description of the patient's general therapy plan. This demonstrates only what the patient has to do in each specific case, but it does not tell us anything about how the patient can reach the necessary result. For example in dynamic aphasia, in a disorder of producing active speech, the following tasks are recommended: retelling a story, telling from pictures, making up sentences from given words, preparing written and oral essays and so forth. An inadequacy of these methods can be seen when the patient is unable to write essays independently and besides he is unable to form a narrative with the help of pictures. Therefore one has to devise methods for the patient which will enable him to perform every exercise. It is absolutely inevitable to develop methods that organize the functioning.

For example in dynamic aphasia the re-establishment of expressive sentences is begun with performing a series of successive operations in which concrete aids are first widely used and which then are gradually dropped out. At first, the programme is performed with the help of a therapist. Later the patient has to perform the whole series of operations independently:

1. Take the picture.
2. Look at it and think about its contents.
3. Divide the picture into logical parts and draw a circle around each part.
4. Connect with an arrow those parts of the picture that are associated together.
5. Check your work.
6. Place a needed amount of cards in position.
7. Say the sentence aloud.
8. Form another alternative sentence.

As optimal therapy methods leading to a permanent rehabilitating effect, such methods are considered that re-establish the inner structure of the disturbed factor in the function by externalizing special operations whose successive, multistage performance can lead to the reorganization of the disturbed function. Corresponding series of operations form a programme that will gradually guide the patient to the disturbed function and will give him a possibility to control this function. At first, this kind of correctly composed programme serves as a guide to re-establishing the function (orientation phase). Later, when the patient has some command of the programme, he can use it in order to adjust the performance of different kinds of

functions himself. It is especially important in therapy that the programme, which first affects the function to be re-established, will lead to creation of such a programme that the patient can organize himself and he can have some effect on. The rehabilitation will not be finished until the execution of the disturbed function is transferred to the patient's control and the function is going actively on.

A function that goes on automatically is analyzed as a series of successive operations. These are accessible to the patient and they function as external control at first. Later, their execution will serve as a means of self control. In this way, the patient is given a chance to cope with the disturbed function already at the beginning of the therapy.

By adopting a stable signal system we will guarantee the necessary reanalysis and it will also increase the effectiveness of the re-establishment of functions that are guided from the outside. This proves that there will take place continuous feedback in a psychic function, a comparison between the performed and intended functioning as well as simultaneous correction of errors. The principle of feedback presupposes special means that makes the patient easier to control his own functions. For example, such tasks as using a mirror which makes it easier to control the re-establishing of movement and speech functions, or a tape which gives a chance to compare one's actual speech with the goal to be attained, have a principled importance in these conditions.

This type of therapy does not use specific methods isolated, because they could lead only to temporary or partial result. Instead, it uses the whole system, whose methods are simultaneously subject to serving the general tasks and directed towards resolving the actual therapy tasks.

Special, separately used methods can overcome only one component of the defect, but they do not re-establish the psychic process as a whole. The methods that are used here presuppose a multidimensional analysis of the defect. They will solve tasks that re-establish the function as a whole. All methods that belong to the system, are directed to solve a separate task. The method system has to suit the nature of the defect and, above all, be directed to overcome the principal defect. In that kind of systems there is often one primary method around which the others are grouped. Nevertheless, all of them function together to solve the task as a whole. Every method system will further include a series of tasks and exercises which strengthen the system effect. For example in sensory aphasia, where the immediate discrimination of phonemes is impossible, the rehabilitation must be directed to incorporation of phonetic identification process to a new functional system. The

rehabilitation of speech understanding is based on the organization of phonemes, where the immediate identification of phonemes is replaced by a detailed series of operations. These series are supported by kinesthetic and visual analysers and by the abstract meaning of phonemes. The operational series are the kernel of the whole multistage rehabilitation process of sensory aphasia.

The models of rehabilitation, their methodic constituents and the development of their psychological basis can be realized only with the help of the established method system, not by searching methods to be used separately. From the point of view of rehabilitation the progress is built upon the external guiding of the concrete functioning. With the help of this guiding there will arise some internalization of given knowledge and the automatization of performance which is to be re-established.

Next I will examine the psychological nature of the contents of the rehabilitation programme. Rehabilitation programme has different kinds of demands. As distinguished from normal therapy, in rehabilitation therapy, what is functioning only, is the functioning of the disturbed factor in the function, but not all the connections of the functions. The undisturbed factors support the realization of the performed programme, in other words, a series of successive operations. For example if a patient's defect is in reading at the level of optical identification of letters, the programme has to be planned so that there will be reserve functions that compensate the immediate optical identification of letters with another method. The other factors in the reading process are not the targets of rehabilitation and therefore they are not shown in the programme. The contents of the programme depends on the structure of the defect and therefore is defect specific every time.

One of the most important demands of every programme is that contents of the programme will be carefully chosen. The programme has to take into consideration the multistage nature of the function to be re-established and no operation can be excluded. If even one operation is excluded, a successful re-establishment of the function is not any more guaranteed. A selection of the programme's composition, a succession of operation's tasks and unlimited repetition are the criteria that can lead to the patient's high activity and independence in order to overcome the defect.

To have success in the rehabilitation of the disturbed psychic function we will need a construction of adapted operational series whose goal is the right performance of the final task. Also conditions that enable the patient to adopt externally the given programme in order to perform the disturbed function and to learn to control it later, have to be created. Within the limits of

rehabilitation there will also arise the problem of creating a method of internalization of the function model. This question is not even stated in the literature or it is only referred in passing that the method will be integrated. Nevertheless, the mechanism of integration remains unknown. The function model given to the patient can arise, when the area of operations diminishes or when individual operations brought from the outside and external aids diminishes and the performance of function is transferred gradually from one level to another.

As a theoretical and practical basis of methods we can use a theory developed by Leontyev and Galperin (Leontyev 1977 and Galperin 1979). One thesis of the theory is that any knowledge or skills cannot be transmitted as such. A learner can adopt, only when he himself has to master of performance of a given operation system. Therefore, consistency has to be taken closely into consideration in rehabilitation. Shortening of a function that has been formed too early will present difficulties.

If we want impressions of external linguistic aids can be found in patients who have received a long period of rehabilitation and we also find signs of fact that the restored function includes unformulated features that served as means in the material level of realization of the process. Therapy that includes concrete tasks in order to restore different functions has above all to be released from the general tasks of rehabilitation. In order to restore the function successfully, one has to correct the function model. The therapist has to transfer, move away disturbing mechaniams with the help of this function model that guides and organizes the patient's activity. This is an important principle in rehabilitation.

The goal of all methods that are directed to rehabilitation of the main defect is the restoration of speech as a unitary higher psychic function. The methods used here express a comprehension of speech as a complex but coherent system where all levels and components are connected with each other. Therefore, these methods also take into consideration the use of verbal communication that takes place at the higher speech level. This covers activity, the knowledge of language, the restoration and understanding of language, extending to the connections between words and things.

The specific feature of the present set of methods is that every method has to come up with two requirements: A therapy of the concrete defect and the secndary disturbance has to restore speech as a whole system, and the methods have to be systematic. The methods are not used separately but as one set of methods, which as

a whole is directed to the therapy of the corresponding defect. Specific methods can be used in many different systems, for example, nonverbal working models can be used with other methods in the therapy of spatial defects. In other cases they arouse involuntary forms of speech and in other cases they control non-productive speech.

The rehabilitation of speech and speech process is carried out in a wide area, as a unitary speech system that has been damaged in some way when the given component of the structure has been disturbed. The specific components need some compensation and others are taken along in order to support the work. Only with the help of that kind of methods that take into consideration the structure of the defect and its systemic effect on all the conditions of speech forms can the disturbed balance of the internal speech system and the outer communication system be restored. The principal leading methods are nevertheless always different and depend on the mechanism which lies behind the disturbed speech.

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SEMANTIC APHASIA AND LURIA'S NEUROLINGUISTIC MODEL

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1. INTRODUCTION

In a research project on assessment of aphasia different types of test tasks are evaluated with regard to their diagnostic value. A diagnostically valuable test task should ideally i.a.

- reveal possible deficits
- differentiate between different types of aphasia
- provide the basis for the setting up of a rehabilitation programme.

The present project includes test tasks using the dichotic listening technique and the visual half field technique but also conventional types of tasks i.e. spontaneous speech, repetition, naming, sentence and story comprehension. This paper only deals with the sentence comprehension task.

When constructing the sentences for the sentence comprehension task I was influenced by Luria's writings on this type of items (Luria 1976). That is why I paid special attention to items expressing logical-grammatical relations. I followed Luria's own examples and used sentences expressing comparative relations like "bigger/smaller than" and temporal relations as in the sentence "He went shopping after he had washed the dishes. What was he doing first?". According to Luria this type of sentences is extremely difficult or impossible for patients with semantic aphasia to handle.

2. SEMANTIC APHASIA

Semantic aphasia will follow a lesion of left hemisphere parieto-temporo-occipital cortex.

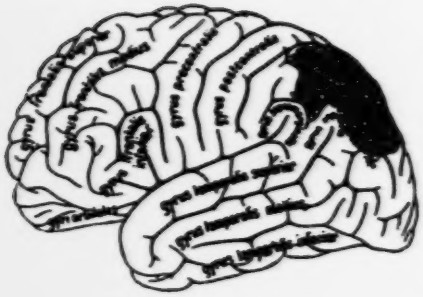

Figure 1. Approximate site of lesion in semantic aphasia as suggested by Luria.

The syndrome of semantic aphasia is described by Luria (1976, 1980) according to the patients' test performance as follows:

- fluent spontaneous speech with word finding problems and verbal paraphasias
- in dialogue there are problems with the understanding of complex grammatical constructions and especially those expressing logical-grammatical relations
- repetition of single words and simple phrases in unaffected but complex phrases are almost impossible
- naming is difficult and gives rise to verbal paraphasias. Prompting helps.
- reading is difficult. The patient cannot understand complex phrases. Some patients cannot get the words unless they try a letter by letter strategy.
- in writing there are problems associating the phonemes with the corresponding visual symbols, graphemes.

Of these symptoms Luria emphasizes the problems with understanding and use of complex grammatical constructions and the instability of lexicon although understanding of isolated words far exceeds the ability to handle complex grammar. Luria refers to a basic difficulty in performing spatial analysis of language. The lesioned zones (see Fig. 1) are indispensable for a complete analysis of incoming external information. These zones "combine successively arriving visual, tactile, auditory and vestibular stimuli into a single simultaneous schema, i.e., they carry out the simultaneous (spatial) synthesis of this information" (Luria 1976: 196). For verbal material this means "fitting incoming lexical elements into a single simultaneously surveyable logical-grammatical (quasi-spatial) structure" (loc.cit.).

3. METHOD

3.1. Patients

In my own project which has a case study design I have 8 patients, 4 men and 4 women. Their lesions differ in etiology and location. The lesions are at least three years old and consequently in a chronic state. (Etiology and lesion for each patient is shown below, Figs. 5-12.)

The patients have all been tested with different types of tasks and the traditional aphasia assessment was videotaped. The evaluation of these tests will not be discussed here. We will deal only with the sentence comprehension task.

3.2. Procedures

Some of the sentences were always presented together with a picture. Examples of this kind of sentence are the following:

A. THIS IS THE ELDER SISTER AND HER LITTLE BROTHER.
B. THE CAR IS SMALLER THAN THE BALL.
   THE CHAIR IS BIGGER THAN THE TABLE.

The sentence could be right or wrong in relation to the picture and the patients were to answer "yes" or "no" in reply to the statement given.

Figure 2. Picture shown with sentence A.

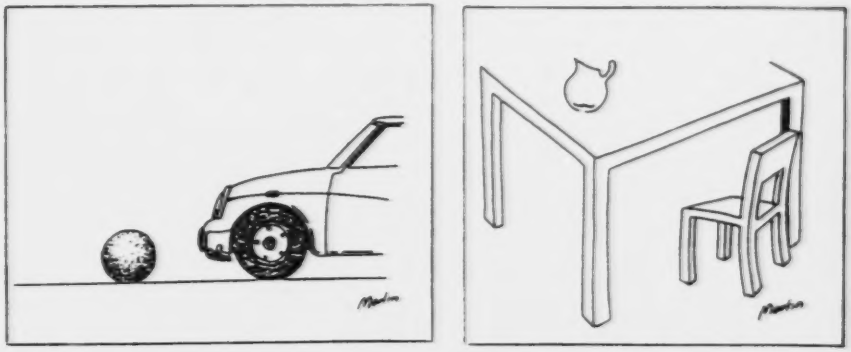

Figure 3. Examples of pictures shown with type B sentences.

Other types of sentences are these:

C. MY FATHER'S SON IS MY BROTHER. RIGHT OR WRONG?

D. HE WENT SHOPPING AFTER HE HAD WASHED THE DISHES. WHAT WAS HE DOING FIRST?

The latter type of sentence (D) requires more than yes/no-answer and patients without oral speech are offered pictures for a pointing response.

As a control there are also sentences such as:

E. FIRST HE WENT SHOPPING AND THEN HE WASHED THE DISHES. WHAT WAS HE DOING FIRST?

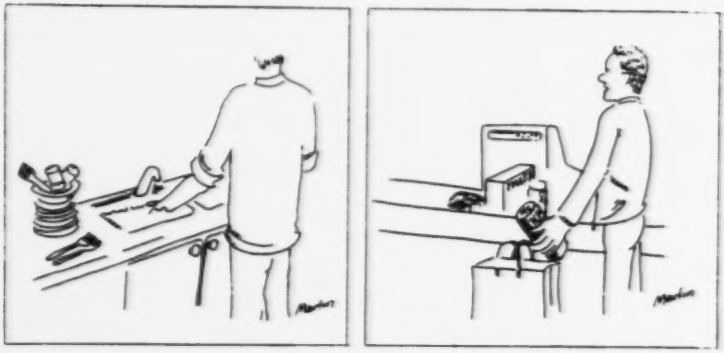

Figure 4. Pictures shown with sentence of type D and E for pointing response.

In all sentences I have used simple well-known words in order to stress the grammatical structure and not the lexicon. The testing procedure was characterized by Luria's philosophy about aphasia assessment. This means that test items are not only given and the result scored but there is a kind of dialogue between therapist and patient about the items and it is the outcome of this dialogue that determines scoring and not the very first answer from the patient. Even the patient is supposed to learn something about his language capacity during assessment. Doing it this way we used about 10 minutes for 15 sentences for this test.

The patients' ability to handle spatial relations expressed by prepositions was tested through the Token Test whose part V contains such items.

4. RESULTS

Table I shows the results summarized in plus or minus signs. The plus sign means that all sentences were correctly understood or that the patient failed the first one when a new type of sentance was introduced. Some patients need what might be termed 'a soft start' when facing new tasks and evaluation of cognitive flexibility was of secondary interest here. The brackets mean that the patient was able to understand the sentence with minimal help from the administrator of the test.

Table I. Performance on sentance comprehension task for 8 patients.

	AK	EG	HL	SN	BE	HA	NG	LA
The elder sister and her little brother	+	+	+	+	+	(+)	-	+
...bigger than... ...smaller than...	+	-	+	+	-	+	-	+
My father's son is my brother	-	-	(+)	+	+	+	-	-
He ... after he had ... What did he do first?	-	-	-	+	-	-	-	-
First he ... and then he ... What did he do first?	+	+	+	+	+	-	+	+

Brackets ( ) indicate that the patient needed some help to succeed with the task.

It can be seen that all patients except SN were impaired. Patient SN has a right hemisphere lesion and no aphasic problems.

Figure 5. Pt. SN, male, age 64. Hemorrhage(s) at age 60 (and 61?). Lesion traced from CT scans.

One of the patients, NG, had problems with all the items. She has a large left hemisphere lesion.

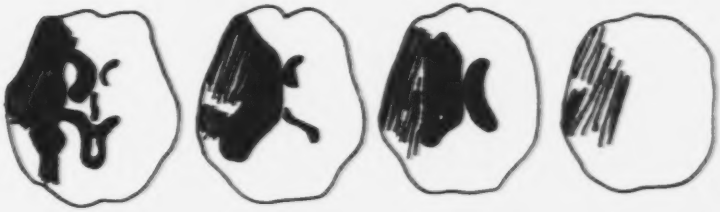

Figure 6. Pt. NG, female, age 55. Op. hemorrhage at age 34. Lesion traced from CT scans.

Patient EG's results were similar to NG's although he is slightly better. He has right hemisphere lesions with aphasia although he has always been a right-hander.

Figure 7. Pt. EG, male, age 64. Op. head trauma at age 55. Lesion traced from CT scans.

The results of AK and LA were rather similar. They both managed the sentences with adjectives expressing one relation but failed on the sentences where the grammar of syntax is crucial for understanding. Their lesions, however, differ with respect to site and etiology although there is overlapping of lesion location.

Figure 8. Pt. AK, male, age 62. Infarction at age 59. Lesion traced from CT scans.

Figure 9. Pt. LA, female, age 38. Hemorrhage at age 30. Lesion traces from CT scans.

Patients HL and HA had similar results although they needed some help with different types of sentences (indicated by brackets in Table 1). Their lesions are certainly not identical but there is a great deal of overlap as regards lesion location.

Figure 10. Pt. HL, male, age 41. Infarction(s) at age 33 (and 35?). Lesion traced from CT scans.

Figure 11. Pt. HA, female, age 64. Op. tumor at age 51. Lesion traced from CT scans.

Patient BE, finally, had problems with comparatives like "smaller/bigger" and temporal relations as expressed in Type D in sentences. She has a lesion along the central sulcus including Broca's area.

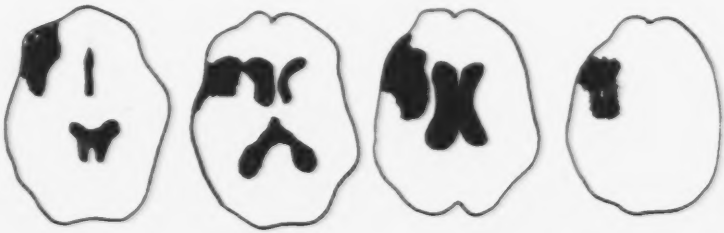

Figure 12. Pt. BE, female, age 64. Infarction at age 59. Lesion traced from CT scans.

Only two patients, SN (non-aphasic) and HA, passed the prepositional items of the Token Test without trouble. As can be seen from Table II they also had the highest total score on part V.

Table II. Performance on the Token Test, part V, which includes 6 items with prepositions.

	Total score	Prepositions
AK	6/21	4/6
EG	-	-
HL	9/21	5/6
SN	17/21	6/6
BE	6/21	6/6
HA	18/21	6/6
NG	5/21	3/6
LA	12/21	5/6

To summarize, comparatives were easiest for the patients to understand while spatial relations were more difficult and temporal relations, expressed as they were in the items used in this study, were almost impossible to handle.

5. DISCUSSION

The conclusion from these data can only be that Luria's writings about semantic aphasia and the processing of sentences expressing relations have to be modified. The sentences used in this study are exactly comparable to Luria's own examples and all the aphasic patients irrespective of lesion site were impaired especially on sentences where grammar governs syntax. In these sentences the grammatical markers are clearly abstract.

In sentences of Type A and B (older/bigger/smaller) you may easily picture the different concepts for yourself and comparatives like "bigger" and "smaller" might be processed like lexical units and in the word order given. However, in sentences like Type D (HE WENT SHOPPING AFTER HE HAD WASHED THE DISHES) it is not sufficient to understand "shopping" and "wash the dishes" because the words "after he had" are of great importance and moreover, they "destroy" syntax which has to be understood "backwards". This kind of temporal relation was difficult for all the aphasic patients.

The understanding of spatial relationship through prepositions is hard to evaluate from this study. According to results of the Token Test all the aphasics except HA had some difficulties with the understanding of prepositions. Only HA performed without errors but, on the other hand, none of the patients failed on all items. It is possible that some patients found the lexical units of the sentences (e.g. "black circle, white rectangle") slightly unfamiliar and thus hard to understand. This would lead to poor performance on this task. As already mentioned, the sentences of my own test only contain highly familiar words.

In 1980 Hier et al. published an article entitled "Semantic aphasia: A neglected entity". They presented three cases diagnosed as semantic aphasics. Hier et al. emphasized that all three cases were impaired in their understanding of grammatically complex constructions and that they had been especially examined with regard to the understanding of sentences expressing different kinds of relations.

Hier et al. found that the patients' understanding of comparatives was good. This result is supported by the study in this paper. Luria postulated that the understanding of comparatives is impaired in semantic aphasia. Hier's findings do not support Luria in this respect. Understanding of passive sentences and sentences expressing spatial and temporal relations was impaired in Hier's patients.

Of the three patients in Hier's study one had bilateral lesions and it seems unwise to refer to such a case. The only data on lesion location for the remaining two cases are early CT scans from the first week after stroke (infarction). These scans show lesions of about the same area as Luria suggested for semantic aphasia. It is far from likely, however, that the lesion area is reliably shown in the first week after a stroke.

Even though Hier's data seem to confirm Luria's description of semantic aphasia symptomatology and lesion location (at least for two of the patients) it is a serious shortcoming that patients with different lesion location and different aphasic syndromes have not been given the same tasks.

In several works Luria presented what might be called a neurolinguistic model based on a more general neuropsychological model. His ideas about brain-language relationship are often described in close connection with clinical findings (Luria 1970, 1976, 1980).

Luria emphasized that all mental operations are the result of a functional system where different parts of CNS are contributing to different aspects (or qualities) of mental activity. When referring to language behaviour he made a front/back dichotomy where the anterior part of the brain was thought of as being

responsible for syntagmatic organization of language and the posterior part for paradigmatic organization. Syntagmatic organization should be seen as the application to language of an underlying basic psychological ability for the programming, regulation and verification of behaviour. Paradigmatic organization reflects an underlying basic psychological ability for integration and spatial analysis of incoming information. Luria claimed that complex grammatical structures and especially those expressing relations, which certainly have a "spatial" quality, are mainly dependent upon intact posterior tertiary areas of the left hemisphere.

Although Luria strongly emphasized that language behaviour always requires syntagmatic as well as paradigmatic organization he claimed that a qualitative analysis of aphasic language can reveal which aspect of language organization has been most impaired. When describing different aphasia syndromes Luria used concrete examples of test performance to show how different syndromes affect different aspects of language. As regards semantic aphasia the present study shows that understanding of sentences expressing relations was impaired in all aphasics irrespective of lesion site. Perhaps a more refined analysis of the performance could reveal interindividual differencies. It is hard to believe, however, that any specific brain area could be of special importance for understanding of such a complex sentence as "HE WENT SHOPPING AFTER HE HAD WASHED THE DISHES". It seems more likely that syntagmatic and paradigmatic processing must be almost equally involved. It is in a way contrary to Luria's own writings about brain language relationships that one brain area (or region) should be more important than others for processing of such complex grammatical structures. The only reasonable dichotomy concerning brain function in this case could be a left/right one. Most probably it was for educational reasons that Luria made the descriptions of his aphasia syndromes so clear-cut that he comes close to the position of a true localizationist which certainly is in contradiction to himself.

Let us now go back to the beginning of this paper and the criteria for diagnostically valuable test tasks. The first criterion to reveal possible deficits seems to be met in that all the aphasics had some difficulties with the task, whereas the non aphasic patient did not. What remains to be shown is whether the difficult sentence (Type D) really can be understood by the average person! The second criterion, differentiation between different types of aphasia, has not been met. Patient AK and LA showed exactly the same pattern as regards understanding but their total clinical picture differs widely.

This leads to problems with the third criterion. If the task does not differentiate between different types of aphasia, it gives no clue about the origin of the defect and hence very little guidance for rehabilitation. Perhaps it is futile to expect that a task involving comprehension of "spatial" grammar should differentiate between different types of aphasics? The more complex the task the less probable it should be to find specific brain correlates to behaviour.

In order to reveal a true spatial deficit it would be wise to start with simple tasks and confine oneself to a greater number of sentences with e.g. comparatives and spatial relations. To minimize the effect of very special individual processing strategies a large group of patients should be examined.

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APHASIC SPEECH ERRORS AND THEIR LINGUISTIC INTERPRETATION

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1. INTRODUCTION

The aim of this paper is to make more explicit the tacit assumptions about the nature of language and aphasia that lie behind different theories about these phenomena. Especially I'm going to discuss - from a rather critical point of view - the hypothesis based on structural and generative linguistics in "explaining" the behavior of aphasics. One of the important points is the difference between spoken and written discourse. The same problems will be discussed at length in my licentiate thesis (Kukkonen, forthcoming). The examples at the end of the paper are discussed also by Kukkonen and Pajunen (1985).

2. THE PROBLEM

The idea of using linguistic theory in interpreting aphasic speech errors goes back at least to the 19th century to Steinthal, the pupil of Humboldt and Herbart (Kotten 1972: 9, Ivić 1966: 50-53). The tradition of using errors in linguistic performance as evidence for the construction and testing of hypotheses on language and language behavior is as old as that. Paul and Delbruck were among the first forerunners in this field (Fromkin 1980: 1). Yet, the collaboration between aphasiology and linguistics has not been as intimate as one might wish.

The main syndromes of aphasia have been rather established in their principles already before the time of Broca, i.e. the middle of the 19th century (Benton 1982). Also the classification of the main types of errors in aphasic speech is considered to be rather established by now. I shall offer a cursory classification of aphasic speech errors from a linguistic point of view. This classification intends to be as neutral as possible as concerns the different theories or "explanations" of these errors.

1. "phonemic" errors in speech production
2. "phonemic" errors in speech perception
3. paraphasias based on the sound structure of the word
4. jargon
5. semantic paraphasias
6. difficulties in naming
7. difficulties in understanding the words
8. grammatical mistakes
9. disturbances of metalinguistic knowledge (i.e. knowledge of language)

The classification as such is not a solution to the problem, and a more profound description of these errors is the case for the linguist.

3. THEORIES OF APHASIA

Certain types of mistakes tend to cluster together in the speech of brain damaged patients. There are some taxonomic studies with objective clustering analysis (e.g. Kertesz 1979, 1982). The clustering of different symptoms of aphasia correlates to a rather great extent with classical aphasia syndromes distinguished in different theories of aphasia. The most important schools are the one based on Lichtheim - Wernicke tradition, the globalist school and Luria's theory. The clustering analysis doesn't solve the problems of aphasia syndromes, because the results depend on the symptoms tested and the clustering of the symptoms says nothing about the origin of symptoms. They may be independent or they may have a common basis.

Theories of aphasia do not offer any deep-going analysis of the aphasic errors. It's rather common to assume some kind of a 'general faculty of language'. This tradition goes back to Broca. Attempts have been made to differentiate between certain types of language processing. In Lichtheim - Wernicke tradition language behavior is classified according to the task to be performed: speech production, speech perception, repetition etc. Luria has developed a qualitative analysis of language processes and makes a distinction between the marginal speech disorders, secondary and tertiary speech disorders. Even he must admit, that the functioning of language is unknown in its details, as can be seen from the following citation from his description of telegraphic style:

> "In patients with the severest lesions of the anterior speech zones (Broca's area) the picture which I have described elsewhere as "efferent motor aphasia" arises. Such a patient cannot switch from one articuleme to another and he is unable to pronounce whole words (not to mention sentences). By contrast, in patients with speech disturbances on the type under consideration in this section the whole picture of the speech defects is quite different." (Luria 1976: 74.)

And he goes on:

> "This disintegration of the syntagmatic structure of verbal expression, ..., is one of the most fundamental phenomena in neurolinguistics. We still know very little about the mechanisms lying at its basis". (Luria 1976: 76.)

4. LINGUISTIC THEORIES

Different linguistic theories can try to illuminate the nature of the 'general faculty of language' and the way it functions. The first linguistic theory that tried to explain aphasia was that of Roman Jakobson. Jakobson based his work on the principles of structuralism, and his ideas have been seminal to most of later linguistic study of aphasia. The main ideas of European structuralism in linguistics had been expressed by Ferdinand de Saussure. One of the corner-stones of structuralism is the definition of language as "the social side of the speech, outside the individual who can never create nor modify it by himself" (Saussure 1966: 14). According to Saussure, language is never complete in any speaker, it exists perfectly only within a speech community. Language is the norm of speech. Anyway, also Saussure thinks that aphasia results not from a disfunctioning of the various organs of speech, but it is caused by disorders of the more general faculty, the linguistic faculty proper (Saussure 1966: 10-11). Noam Chomsky, the founder of the generative grammar, accepts the Saussurean distinction between langue and parole, or competence and performance, as he calls them. Anyway, he defines competence as the ideal speaker-hearer's knowledge of his language in a completely homogeneous speech-community (Chomsky 1967: 3-4). The main differences between the theories of Saussure and Chomsky are that Chomsky defines competence as a property of an individual, and as a system of generative processes. As concerns the interpretation of Saussure's theory and that of Chomsky's in the study of aphasia, there do not exist any fundamental differences.

The corner-stones of the structuralistic interpretation of aphasia are that one admits the existence of some kind of a general faculty of language in the individual's brain and that the disturbances of this general faculty follow the structure of langue or competence as described by the linguist. In other words, it is thought that the competence models of language are also psycholinguistic models. According to the structuralist-generative tradition, language consists of different components. In the early theory by Jakobson these components were phonology, grammar and lexicon. And indeed, Jakobson (1978: 44) finds three basically different types of aphasia: "Lautamnesie" (sound amnesia), "Agrammatismus" (agrammatism) and "Wortamnesie" (word amnesia). In a severe disturbance the patient has lost the elements (sounds, grammatical morphemes, words) totally, in a milder disturbance only the opposition between the items is disturbed, and they occur in free variation. These milder disturbances are called "Lautparaphasie" (sound paraphasia), "verbaler Paraphasie" (verbal paraphasia) and "Paragrammatismus" (paragrammatism).

After this early work of Jakobson, the linguistic theory has been developed. The components the language system is assumed to consist of are different than in the early forties. The theory by Caramazza and Berndt (1982) seems rather orthodox from this point of view, and the theory of Luria has been developed to the same direction (by e.g. Akhutina 1975). Four or five different types of aphasia are distinguished: phonological aphasia, syntactic aphasia (which may include a morphological disorder, or the morphological disorder may form a syndrome of its own), lexical semantic aphasia and aphasia in which the combination of the meaning of a sentence from its elements is disturbed. I have discussed this theory more thoroughly in an earlier paper (Kukkonen 1984, see also Kukkonen, forthcoming). The problem for the linguists and aphasiologists basing their work on this theory is to find out which component of language is disturbed in every syndrome, and the description and "explanation" of all different symptoms in accordance with the theory. E.g. agrammatism (the best studied syndrome) can be offered different explanations: it may be caused by the disturbance of syntax (Berndt & Caramazza 1980, Akhutina 1975), or, depending on the variety of linguistic theory one chooses, by that of phonology (Kean 1977) or lexicon (Swinney & al. 1980). The most serious problem with this theory is that it accepts the distinction between competence and performance. The model of language processing is divided into two main components, planning and execution, which correspond to the distinction between competence and performance, respectively. According to these theories, the planning phase has suffered in aphasia. In practice, it's difficult to make a clear

distinction between a disorder of planning and that of execution. Competence grammars pay no attention to how people normally communicate in everyday situations.

Linell (1983) gives many examples of spontaneous speech production in informal situations and shows that one begins the execution of an utterance before one has planned in any detail what one is going to say. The linguistic tradition, however, has been concerned primarily with written language. No attention has been paid to the characteristics of everyday speech: false starts, ellipsis, pauses, fillers of these pauses etc.

Recently it has come clear that in order to understand speech behavior one has to abandon many concepts that have emerged in studying written language or formal style. One of these is the phoneme. Many linguists interested in aphasia want to find aphasic syndromes that correspond to disorders of separate components of language, e.g. phonology. What is a phonological disorder like? From the linguistic point of view one would be interested in finding a disorder that affects both speech perception and speech production. A patient having this kind of a disorder should have plenty of literal paraphasias in spontaneous speech and difficulties in tests of phoneme discrimination. It is assumed that speech perception is based on phoneme discrimination. These disorders do not typically cluster together in aphasia, but there is a syndrome in which one could expect this kind of a disorder at the background of the symptoms, namely Luria's afferent motor aphasia that corresponds roughly to what Kertesz calls efferent conduction aphasia. Vinarskaja (1971) has written her dissertation on afferent motor aphasia, and she claims that the syndrome is not homogeneous and, therefore, divides it into three subgroups. This conclusion is based on an experiment where the ability to repeat meaningless syllables, read them and write them at dictation were tested. The patients that are able to repeat syllables but cannot read them or write them to dictation have a phonological disorder. They can repeat syllables, because repetition does not demand phonological analysis - it is only imitation. This analysis of repetition as a cognitive process can be questioned. Caramazza, Berndt and Basili (1983) have used a better series of tests in trying to find a phonological disorder, but they have not analyzed the spontaneous speech of the patient. It is very difficult, if not totally impossible, to differentiate between the co-occurrence of different symptoms and one symptom "of a more abstract nature".

This example shows how difficult it is to find syndromes that correspond to the components of linguistic theories. Indeed, at the state of linguistic art, it's difficult

to drew a distinction between language and other manifestations of cognition on one hand, and between planning and execution on the other hand. Some of the latest achievements in psycholinguistics seem to point to the same direction.

However, most of the psychalinguistic models of speech production and speech perception seem to be based on similar kinds of assumptions as the structuralistic competence theories. One has underestimated the plasticity of language processes and the role of metalinguistic knowledge. For this reason it seems rather fruitless to try to describe the aphasic syndromes as boxes in a model of language processing (see also Linell 1983). Before being able to construct any model of language behavior based on aphasia, one should study the actual language behavior of aphasics much more thoroughly than has been done so far. Further knowledge of aphasic speech errors would surely point out that there are different "normal" strategies by which aphasics try to compensate for their difficulties. The disorder does not point directly to the missing component but to these compensatory strategies.

5. EXAMPLES

Anneli Pajunen and I (Kukkonen & Pajunen 1985) have analysed speech of two agrammatic patients: A has a syndrome of efferent motor aphasia with features of dynamic aphasia; N has efferent motor aphasia with features of acustico-mnestic syndrome and perhaps some afferent motor features. Both patients have difficulties with e.g. deictic elements. There are a couple of deictic verbs in Finnish (e.g. mennä 'to go' and tulla 'to come'; tuoda 'to bring here' and viedä 'to take there'). A and N have difficulties with other deictic elements, too. So, the misuse of these verbs is not simply semantic paraphasia. A's and N's errors are of different kind. N tends to use the proximal variant instead of the non-proximal one: (All names in the examples are changed.)

(1) Hän päätti kirjoittaa yhdelle rouvalle kirjeen että tulisi tänne vierailemaan. Hän sai vastauksen että tulen tänne junalla.
'He decided to write a letter to a lady and ask her to come and visit him. He got the answer that I'll come here by train.'

A, instead, uses the variant that is non-proximal and has lost its deictic nature to some extent:

(2) Tuija menii meille. Tuija ja minä puhuvat hirvesti.
'Tuija went to our place. Tuija and I talk a lot.'

(3) Aamulla minä menen haravoimaan pihaa ulos. Minä menen Arja Saira kello kolme tunnilla. Oulunkylään menen minä 63-bussilla.
'In the morning I go out to rake the yard. I go (to) Arja Saira (at) three o'clock. To Oulunkylä I go by bus.' (Oulunkylä is the place where A lives.)

A also tends to use proper names instead of personal pronouns and personal suffixes. This points to some kind of decontextualisation of A's utterances (the examples here are from A's diary, that is, from a non-canonical speech situation, but same kind of behavior might be observed in canonical situations, too). From a linguistic point of view any far-reaching conclusions shouldn't be drawn on this limited material. The examples here only intend to point out that the aphasics can use different strategies to overcome same kind of difficulties. This speaks against the strictly modular theory of cognition and organization of language.

In English, one of the characteristics of agrammatic speech is the omission of function words. In Finnish there are rather few function words but plenty of suffixes that tend to get omitted or misused by agrammatic aphasics. It's a common observation that the omission of function words is hardly ever total, and e.g. Zurif & Caramazza (1976) have noted that the information value in relation to other words of the sentence is important in predicting which constructions are easy to the patient. We (Kukkonen & Pajunen 1985) have tried to analyze the errors of the previously mentioned two patients from this point of view.

In the languages where the grammatical roles of words or constituents are expressed by word order, as in English, there is no need to pay attention to government. In Finnish these roles are usually shown by case-marking, and that is called government. There is no complete isomorphism between form and meaning in natural language and the government may be semantically or syntactically motivated. By semantic motivation we mean that the case-marking is somehow natural. Our hypothesis is that semantically motivated government is much easier for these agrammatic patients than syntactically motivated government. E.g. mennä 'to go' is one of the verbs of motion. In Finnish local and directional verbs of motion are usually marked rather systematically with local cases and they form a unified lexical field. A uses the verb more simply than is done in everyday speech. 99 % of her sentences have the structure mennä + illative case. A produces 70 % of the sentences correctly. When the sentences get more complicated, A has only 57 % of the sentences correct. Some examples:

(4) Timo minä ja mummu menimme navetaan. (correct)
'Timo I and grand-mother went to the cow house.'

(5) Me menimme Irmalle luo. (correct: Me menimme Irmalle. or Me menimme Irman luo.)
'We went to Irma's place.'

(6) Timo, Toni ja minä menimme retkelle pyörille.
(correct: Timo, Toni ja minä menimme retkelle pyörillä.)
'Timo, Toni and I went to a trip by bicycle.'

In Finnish relation between entities can be expressed by different syntactic structures, and there is no such isomorphism between form and meaning as is case with the verbs of motion. E.g. possessive constructions are difficult for A. Examples:

(7) Me on uusi Taunus. (correct: Meillä on uusi Taunus.)
'We have a new Taunus.'

(8) Äitille on synttärit. (correct: Äidillä on synttärit.)
'It's Mother's birthday.'

It's clear that this alone can't explain all the simplification of grammatical structure in agrammatism. Anyway, the degree of isomorphism between form and meaning is one of the factors that one should take into account when analyzing agrammatism.

I think that finding and establishing factors of this kind is the most important task of linguistic study of aphasia.

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WORD FLUENCY: A TASK ANALYSIS

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1. INTRODUCTION

"I shall say a letter of the alphabet, then I want you to give me as many words that begin with that letter as you can and as quickly as you can. For instance, if I say 'B', you might give me 'bag, battle, bed, and so on'. Do not use words which begin with a capital letter like the names of brands, people, or places such as 'Bob or Boston'. Also, do not give numbers, or begin the same word but with a different ending, for example, 'eat, eating'. Do you have any questions? Begin when I say the letter. The first letter is S."

These instructions cited from Borkowski et al. (1967) describe a typical word fluency (WF) task. Specifically, the test is a phonetic fluency task, going by a name of letter. Another type of fluency measure which the subjects usually find easier, are tests of semantic WF, where one has to name animals, fruits, vehicles and so on within a short period of time (for a description of various WF tests in clinical use, see Lezak 1983).

Originating from intelligence research, WF measures have been used quite widely in neuropsychology. The main reason is that they have been repeatedly shown to be sensitive to frontal lobe damage, particularly to left anterior lesions (Benton 1968, Borkowski et al. 1967, Milner 1964, Perret 1974, Ramier & Hecaen 1970). It is unclear, however, why WF tests discriminate between frontally lesioned and other brain damaged patient groups. Rather vaguely, the defect has been labeled as a "higher-level verbal deficit" or related to the subject's ability to organize his thinking. The single exception is Perret (1974), who suggests that phonetic WF tasks measure ability to suppress the habit of using words according to their meaning. Particularly left frontal damage was related to impairment of this inhibitory mechanism.

In the present paper it will be suggested that models of human problem-solving are useful in the analysis of WF test performance. In problem solving terms, WF tasks would require the subject to define and clarify the conditions of the task (task acquisition), select and employ appropriate strategies, and monitor one's

performance (Fig. 1). It is obvious that a WF task is not a problem in the same sense as tests like the Wisconsin Card Sorting. Still, it can be considered as a "problem" because it represents a new task situation, and the ways of solving it effectively are not always immediately available to the subject. In modern neuropsychology, the frontal lobes have been attributed a crucial role in general problem solving (e.g., Luria 1980, Damasio 1979).

The rest of the paper will focus on preliminary experimental and clinical data of qualitative features in neurological patients' WF performance. It will be suggested that these results could be fitted into the simple information-processing model in Fig. 1, and that one may observe a kind of hierarchy in the susceptibility of these components to neural damage.

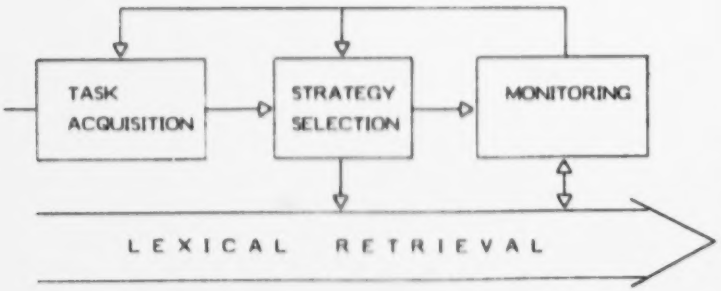

Figure 1. Hypothetical model of word fluency performance.

2. WORD FLUENCY PERFORMANCE IN KORSAKOFF PATIENTS: FAILED MONITORING

This study was performed at the Boston Veterans Administration Hospital under the supervision of Professor Nelson Butters, Ph.D. The WF results are part of a project which focused on the problem-solving skills of chronic alcoholics and Korsakoff patients (Laine 1984, Laine & Butters 1982). (Initial analysis of the data was performed in collaboration with Dr. Elisabeth Moss, Ph.D.)

In one session, several WF tasks were administered to groups of alcoholic Korsakoff patients, chronic alcoholics, and normal controls. Altogether 24 subjects were tested, 6 Korsakoffs, 8 alcoholics and 9 controls. The groups did not differ significantly in age, education or WAIS Vocabulary score (Table 1). We used three phonetic tasks (F,A,S) and four semantic tasks, where the subjects had to list orally as many animals, fruits, vehicles and weapons as they could within one minute. The order of the tasks was fixed starting with the phonetic tests (see Fig. 2). The instructions were similar to those used by Borkowski et al. (1967).

Table 1. Mean age, years of education and WAIS Vocabulary scores of alcoholics, controls and Korsakoff patients.

	Alcoholics (n=8)	Controls (n=9)	Korsakoffs (n=6)
Age, years	56.8 (3.0)	55.6 (2.5)	57.5 (4.8)
Education, years	12.8 (1.8)	11.8 (2.9)	12.5 (2.0)
WAIS Vocabulary scaled score	11.9 (1.9)	12.2 (1.1)	11.8 (3.4)

Standard deviations are shown in parentheses.

The ordinary quantitative results, the number of words produced in one minute, are presented in Fig. 2. One-way analyses of variance failed to reach statistically significant differences between the groups except for the category of fruits. Furthermore, the number of repetition errors or perseverations for each subject was counted. Repetition error is simply an occasion when the subject produces exactly the same word again, e.g., cat-dog-horse-cat. In the total number of perseverations (Fig. 3) the groups differed significantly, and this is solely because of the accumulation of perseverations in the Korsakoff group. The most interesting finding is, that the distribution of perseverations across the tasks (Fig. 4) is highly skewed: in the Korsakoff group, perseverations accumulate to the first task of a test type.

The tentative explanation for the failed monitoring at these points is that here the impaired Korsakoff patient is facing a new task and the limited resources of attentional control available are needed to both task acquisition and strategy selection. One might argue that the severe anterograde amnesia of these patients would explain the high number of perseverations, and short-term memory is certainly involved when the patient attempts to keep track of which words have already been said. Being a purely amnesia-related phenomenon, however, one would expect to find a flat distribution of perseverations. Moreover, a single case of relatively "pure" acute transient global amnesia, which the present author has tested with WF tasks during the attack, did not exhibit perseverative tendencies, although being severely amnesic. Fitted into the model (Fig. 1), the accumulation of perseverations to certain parts of a WF test battery would indicate a Component 3 defect in Korsakoffs. It is notable that various problem-solving deficits have been reported previously in these patients (for review, see Butters 1985). Of course, there still remains a possibility of some kind of an interaction effect between poor memory and defective monitoring, and this question awaits further studies.

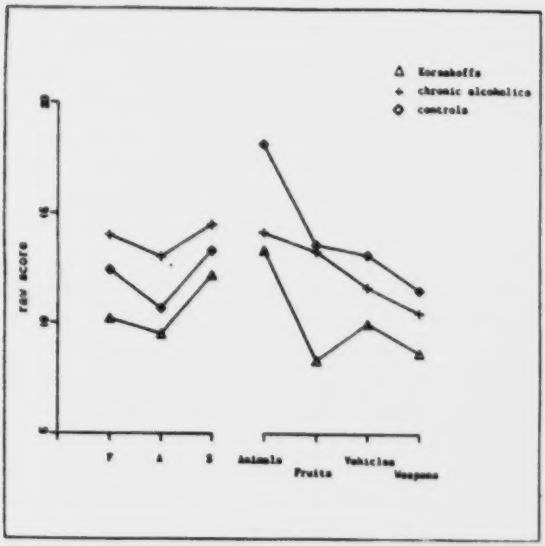

Figure 2. Group means on word fluency tasks.

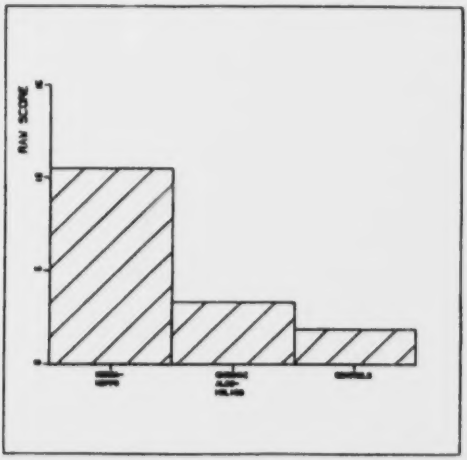

Figure 3. Group means of total perseverations across the word fluency tasks. $F(2,20) = 7.76$; $p<0.01$.

3. DEFICIENT TASK ACQUISITION

Clinical manifestations of the hypothetical Component 1 defect, problems with task acquisition, represent a much more severe disorder often accompanied by even otherwise inadequate WF performance. The data to be presented is based on single cases suffering from a relatively severe "frontal lobe syndrome" defined by neuropsychological assessment. Etiology is variable. Instructions of the phonetic fluency task were somewhat different from the ones used with the Korsakoff patients: only names of people had to be excluded, not names of brands or places.[1]

The first case, patient I.V., is a 71-year-old right-handed female patient suffering from a haemorrhage in the area of the left caudate nucleus. A template of the lesion is presented in Fig. 5. Neuropsychological examination revealed typical frontal lobe damage signs such as planning defects, tendency to perseverate, as well as impulsivity and disinhibition. Moreover, she suffered from amnesia and was also anomic. The results of two WF tasks, naming by letter S and animal listing, are presented in Tables 2 and 3 (see Appendix). On the first task, she shifts to another letter, thus violating the task rules. Also, the sheer number of words produced is quite low. There is one perseverative error. On animal naming, she starts by listing only animals starting with letter K, which exemplifies her difficulties in set shifting. Then she gets stuck on a nonrecognizable word which she does not recognize even by herself! After that, she is being reminded of the task instructions (marked by "!"), and she continues with a couple of animal names starting again with letter K. Towards the end of the task, the interfering effect of the previous phonetic test is once again evident when she starts to produce first names. The final products appear to be totally unrelated words, the last one being a derivation.

The second case, patient S.V., is a 60-year-old right-handed female patient suffering from normal pressure hydrocephalus. The main CT findings were enlarged ventricles as well as decreased density in the frontal and parietal white matter. On neuropsychological testing, a clear frontal lobe symptom complex was observed: she was euphoric, concrete, exhibited some loss of inhibition, and had quite a severe

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1 All of these cases have been examined at the Turku University Central Hospital. Detailed neurological and neuropsychological data of these cases are available from the author upon request.

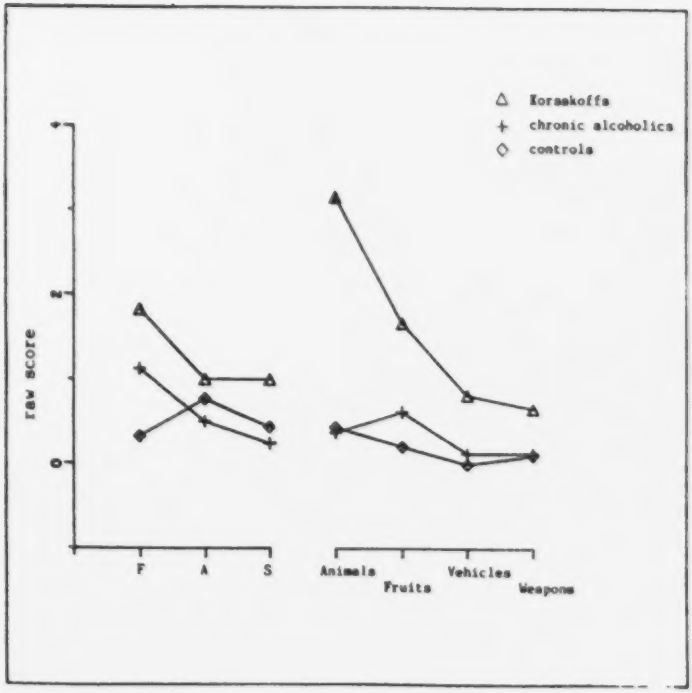

Figure 4. Group means of perseverations on word fluency tasks.

Figure 5. CT scan templates of patient I.V.: Haemorrhage of the left caudate nucleus.

memory disorder. On the phonetic fluency task (Table 4, Appendix), she starts with first names, which she was just instructed not to do. She is reminded of this fact, but there still appears one first name. The next error is a derivation of the word "sugar", followed by a rather uncommon choice: Seven Brothers, being one of the classic works in Finnish literature.

The third case, patient A.E., is different in the way that so called "frontal damage signs" were not so prominent in his neuropsychological status. He is 67-year-old, right-handed, and clinically diagnosed as suffering from senile dementia. His neuropsychological defects were quite extensive, and he had a clear language disturbance with features of fluent aphasia. This kind of a language pathology is typical in Alzheimer's disease (Obler & Albert 1981). On the animal naming (Table 5, Appendix), the patient subsequently produces some paraphasic errors. More interestingly, after the start he soon continues along the lines of the first task abandoning the most recent instructions. At the end, there is one perseverative error.

The WF test protocols of these severely impaired neurological patients show several examples of inadequate task acquisition, i.e., Component 1 defect. A significant part of the errors appear to be related to interfering effects of the previous task. It is notable, that the shifting of a test type (phonetic-semantic) also produced the highest number of perseverations in the Korsakoffs (Fig. 4).

4. FITTING WORD FLUENCY PERFORMANCE INTO A MODEL

Two types of disordered WF performance have been delineated on the basis of the empirical data:

(a) Component 3 defect: perseverative errors

(b) Component 1 defect: breaking the task rules, perseverative errors, poor quantitative performance

The milder, the monitoring defect, can occur in isolation, but the more severe Component 1 defect is accompanied by deficits in monitoring and impaired quantitative performance. The reason for monitoring to impair first can only be speculated upon: monitoring is peripheral to the actual lexical search, and it depends in part on short-term memory. One significant factor in elucidating these pathological phenomena appears to be set shifting. In a way, these shifting or

interference effects can be related to Perret's (1974) notion of suppression of normal (earlier) ways of acting. However, Perret focuses on a single WF test type, and in the present study interference effects are not related to this factor. Rather, other task-related variables such as the order of the tests and the specific instructions seem to affect the form of the set shifting errors.

Although not specifically studied in the present experiments, it is tempting to relate this crude hierarchy of WF pathology to defects in more complex problem-solving tasks. Laine (1984) attempted to describe chronic alcoholics' and Korsakoff patients' defects on 20 Questions Game tasks with a similar model that is applied here. On these difficult tasks, chronic alcoholics had their main difficulties in initially selecting appropriate strategies. The Korsakoffs, on the other hand, used poor strategies throughout the tasks and their performance was not facilitated by the examiner's feedback. This could be considered as a defect in monitoring one's performance. On the basis of preliminary data, it is difficult to conclude whether this monitoring defect is in fact related to Korsakoff patients' perseverations on WF tasks. It can only be noted, that the two Korsakoffs who produced most WF perseverations, were also the most impaired on the 20 Questions Game tasks.

In the present article, the main emphasis has been on error analysis, and Component 2, strategy selection, has not been studied. Potentially, this would give valuable information: Estes (1974) comments that successful performance on these tests depends in part on the subject's ability to "organize his output in terms of clusters of meaningfully related words". Lezak (1983) gives examples of effective clustering strategies of WF tasks. Even though the use of clustering certainly varies from one subject to another, there is one strategic aspect, in which various neurological patients seem to act in a relatively similar fashion. That aspect is the use of "prototypical" names (cf. Rosch 1975) in initiating a semantic WF task. In Fig. 6, the most common first four words in animal naming used by 50 unselected neurological patients are presented. Only three words, "dog-cat-cow", account for nearly half of the total number of counts. It must be emphasized that in this particular WF test no examples of suitable animal names or subcategories were given, and the patient was simply instructed to name as many animals as possible within one minute. As there appears to be unity of performance in this respect, various semantic WF tasks should be useful in prototype research with normal populations.

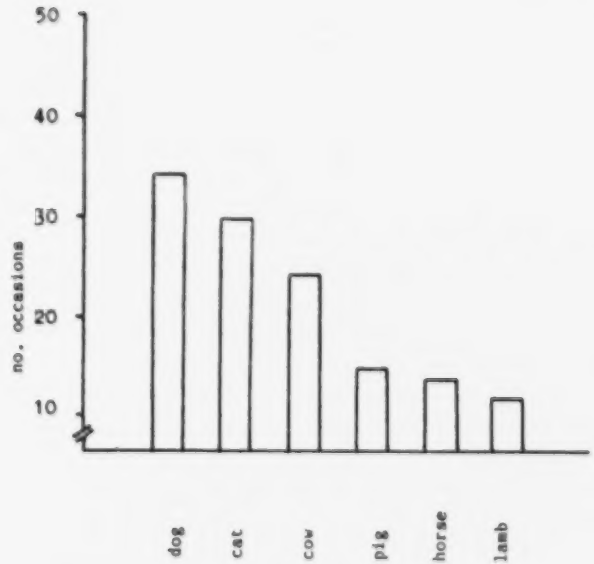

Figure 6. Animal naming: the most common first four words (n = 50).

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Appendix, Tables 2-5. Word Fluency Protocols of Patients.

TABLE 2.

PT. I.V.: WORD FLUENCY BY LETTER S

0 - 15"	SENTTI	CENTIMETRE
	SYSMÄ	(A PLACE NAME)
	SYYHELMÄ	ITCH
15 - 30"	SENTTI	(PERSEVERATIVE ERROR)
	SIELU	SOUL
	SOLU	CELL
	SOPPA	SOUP
30 - 45"	SULHO	BRIDEGROOM
	HOME	MOULD
	HOUKUTUS	TEMPTATION
45 - 60"	-	

Errors underlined.

TABLE 3.

PT. I.V.: ANIMAL NAMING

0 - 15"	KARHU	BEAR
	KETTU	FOX
	KURKI	CRANE
	KUUSYY, EI SE OO	(NONWORD), I GUESS IT DOES
	VISSIIN MITÄÄN	NOT MEAN ANYTHING
15 - 30"	(I) ELÄIMIÄ JUU	OH YES, ANIMALS
30 - 45"	KIURU	LARK
	KOTKA	EAGLE
	KALLE	(FIRST NAME)
	PEKKA	(FIRST NAME)
	PELLE	(FIRST NAME)
45 - 60"	PUU	(TREE)
	HEITTO	THROW (noun)
	HEITTONARU	LASSO

Errors underlined.

TABLE 4.

PT. S.V.: WORD FLUENCY BY LETTER S

0 - 15"	SANNI	(FIRST NAME)
	SUOMA	(FIRST NAME)
	(!)	
	SIRKKA, NE ON KAIKKI IHMISTEN NIMII	(FIRST NAME), THEY ARE ALL PEOPLES' NAMES
15 - 30"	SOKERI	SUGAR
	SIIRAPPI	SYRUP
30 - 45"	FARINSOKERI	BROWN SUGAR
	SEITSEMÄN VELJESTÄ	SEVEN BROTHERS
45 - 60"	SIKA	PIG
	SORSA	WILD DUCK

Errors underlined.

TABLE 5.

PT. A.E.: ANIMAL NAMING

0 - 15"	KOIRA	DOG
	KANA	HEN
	HEVONEN	HORSE
	SIILI	HEDGEHOG
	SIMPPU	COTTUS, A FISH
	SIIRIÄINEN	(SURNAME)
	SAMMAL	MOSS
	SUKSET	SKIS
	SAUVAT	SKI-STICKS
	SAJAT	(NONWORD)
15 - 30"	SURVIAINEN	MOSQUITO
	SAKSANMAA	GERMANY
	SIPPO	(FIRST NAME)
30 - 45"	SAMPO	(FIRST NAME)
	SYLKYLÄINEN	(NONWORD)
45 - 60"	SORSA	WILD DUCK
	SAUKKO	OTTER
	SIILI	(PERSEVERATIVE ERROR)

Errors underlined.

SPEECH AND LANGUAGE DISORDERS IN A DIALYSIS ENCEPHALOPATHY PATIENT AND THE EFFECT OF DESFERRIOXAMINE AND REVERSE-OSMOSIS WATER TREATMENT

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1. INTRODUCTION

Dialysis encephalopathy (earlier also called "dialysis dementia") is a progressive neurological disorder that occurs after long-term hemodialysis in some patients with chronic renal failure. It was first described by Alfrey & al. (1972) and is the leading cause of death in some renal centres. However, its geographical distribution is very varied, so that in some centres the disease has never occurred (Parkinson & al. 1979). It is characterized by a unique speech disorder, seizures, EEG changes, myoclonus, dyspraxia and dementia (Lederman & Henry 1978). Speech and language disorders are often mentioned as the first signs of dialysis encephalopathy (Madison & al. 1977, Barron & al. 1980).

There has been much speculation about the etiology of dialysis encephalopathy, and several causative factors have been suggested, e.g. amino-acid depletion, vitamin deficiency, slow virus infection, abnormal cerebrospinal fluid dynamics, phosphate depletion, a primary seizure disorder, disturbed dopamine metabolism, and disturbances in brain amine neurotransmitter metabolism (O'Hare & al. 1983). Recently, Sweeney & al. (1985) have suggested that a deficiency of a neurotransmitter called GABA might be the immediate cause for dialysis encephalopathy. At present, however, the most widely accepted cause is thought to be the accumulation of aluminium in the cerebral cortex (Davison & al. 1982; O'Hare & al. 1983; Wills & Savory 1983).

The disease was first described in 1972, but no special treatment was invented during the early 1970's; among the treatment methods suggested at that time were e.g. increased dialysis, renal transplantation and various medicines, especially diazepam. But these appeared to have only a temporary effect (Lederman & Henry

1978). In fact, we have seen two patients who developed the symptoms of dialysis encephalopathy shortly after a successful renal transplantation (Lehtihalmes 1984). The mean duration of clinical course until death has been about six months (Chui & Damasio 1980).

As the accumulation of aluminium in the brain became to be suspected as the cause for dialysis encephalopathy, new methods of treatment were suggested at the turn of the 1980's in order to lower the aluminium concentration in the nervous tissue. Since most of the aluminium accumulated in the brain originates from the water used in hemodialysis and penetrates the blood-brain barrier (Banks & Kastin 1983; Schreeder & al. 1983), it has become necessary to lower the aluminium concentration of the dialysis water with different methods, the most efficient of which has proved to be reverse osmosis (Bok & Grondin 1980; Davison & al. 1982). Deionizing the dialysis water has also been effective (Barron & al. 1980). An additional source of aluminium may be the phosphate binding gels (aluminium hydroxide) taken orally by hemodialysis patients in order to prevent hyperphosphatemia which arises in chronic renal failure. This is why excessive dosages of phosphate binding gels should be avoided, especially with children (Alfrey 1985). Recently it has been shown that desferrioxamine has a good therapeutic effect in dialysis encephalopathy (Milne & al. 1983). It seems to mobilize the aluminium from the tissue to the serum, thus easing removal by hemodialysis (Wills & Savory 1983).

2. COMMUNICATION DISORDERS IN DIALYSIS ENCEPHALOPATHY

In the literature four studies can be found on speech and language disorders in dialysis encephalopathy (Rosenbek & al. 1975; Madison & al, 1977; Baratz & Herzog 1980; Barron & al. 1980). Except for the one by Barron & al. these are case studies and do not contain a systematic analysis of the development of speech and language disorders during the progression of the disease. In these studies the following characteristics have been described as typical of dialysis encephalopathy.

Dysfluency has been consistently mentioned as the most striking feature at the early stages of the disease. Various stuttering-like phenomena have been observed: repetitions of sounds, syllables, words, and sometimes phrases, hesitations, difficulties in initiating phonation and articulation, perseveration of stressed syllables and words, inappropriate pauses within and between words, and even

intermittent mutism. In general, the speech rate tends to be very slow, and the speech has a very laboured quality. Rosenbek & al. (1975) also report significant abnormalities in diadochokinesis in their patient. Certain disturbances in prosody could also be interpreted as signs of dysfluency: the patients often use an equal stress on every syllable, and their speech has a staccato-like rhythm ("scanning speech"). The "stuttering" is more marked at the early stages of the disease than terminally. The severity of the dysfluency has been shown to fluctuate increasingly towards the terminal stage of the disease, when it alternates between total mutism and hyperfluency (Madison & al. 1977). The classification of this symptom as stuttering has been criticized, and e.g. Rosenbek & al. (1975) and Barron & al. (1980) prefer calling it either dysarthria or dyspraxia (or a mixture of both).

Another disorder that is often mentioned is irregular articulatory dysfunction, which includes imprecise consonants, sound substitutions and omissions with frequent attempts at self-correction. The articulatory movements are irregularly and unpredictably deviant, and the errors tend to increase as sentences become longer. The nonverbal oral-motor functions are also inaccurate and uncoordinated. The articulation errors tend to be phonetic rather than phonemic.

Thirdly, disorders of phonation and prosody have occurred. The type of these disorders, however, seems to be individual to a great extent. The phonation quality has been described as varying from a strain-strangle dysphonia in some patients to breathy phonation in others. The pitch is often monotonous and lower than normal with occasional pitch breaks, especially in conversational speech. In some patients, on the other hand, uncontrollable rising and falling glides in pitch can be heard.

In addition to the above mentioned disorders of speech, various language deficits are sometimes observed. Auditory comprehension has been described as normal or mildly disturbed. The difficulties become more apparent as grammatical and semantic complexity increases. Some mild word-finding problems and paraphasias may be observed at the early stages, but clearer indications of aphasia appear towards the terminal stage. In addition, there are also aphasic disorders in writing and reading.

More generalized neuropsychological disturbances in dialysis encephalopathy have also been mentioned. These include deterioration in memory function, reduced attention span, apraxia, and diminished interest in vocational, family and social activities. Even clear signs of mania have been observed (Jack & al. 1983). With the progression of the disease these symptoms become more severe, and at the final

stages patients develop generalized dementia with disturbed thought processes, mental confusion, delusions, hallusinations and disorientation.

3. THE PRESENT CASE

The patient is a 48-year-old female factory worker who developed uremia after chronic nephritis in the year 1978. Hemodialysis was begun the following year, three times a week. In summer 1981 she had some mild paroxysmal speech difficulties after dialysis. In summer 1982 after dialysis she had a sudden period of mutism which improved after half an hour. She had also some pains in her ribs and mild mental slowness was observed the following autumn, and the diagnosis of dialysis encephalopathy was made. The aluminium concentration of the tap water in Helsinki is very high and therefore the purification of the dialysis water with reverse osmosis was started. Desferrioxamine (Desferal®, Ciba-Geigy Co. Switzerland) treatment was also started in autumn 1982; the desferrioxamine, 2 g in 100 ml normal saline, has been infused to the venous line of the dialyzer during the last hour of the hemodialysis session once a week.

Brain CT scan was normal in autumn 1982 except for mild atrophy. In spring 1984 an NMR investigation was made and it showed some abnormalities adjacent to the left occipital horn. These changes were taken to be related to encephalopathy. The EEG recordings were taken every six months and some progressive abnormalities were observed (slow wave activity, especially in the left temporal area) at the successive recordings.

During the period of this study a speech pathologist assessed the patient five times, always the day following dialysis. The methods used were the Frenchay Dysarthria Assessment (Enderby 1983; see Fig. 1), the Token Test (De Renzi 1977), a test for word fluency (words per minute) and an assessment of reading and writing abilities. In addition, a neuropsyhological investigation was performed by a psychologist.

The equipment used in the instrumental analysis consisted of a Kay Elemetrics Sona-Graph 6061B and a microprocessor-based digital Speech Processing System (SPS-02).

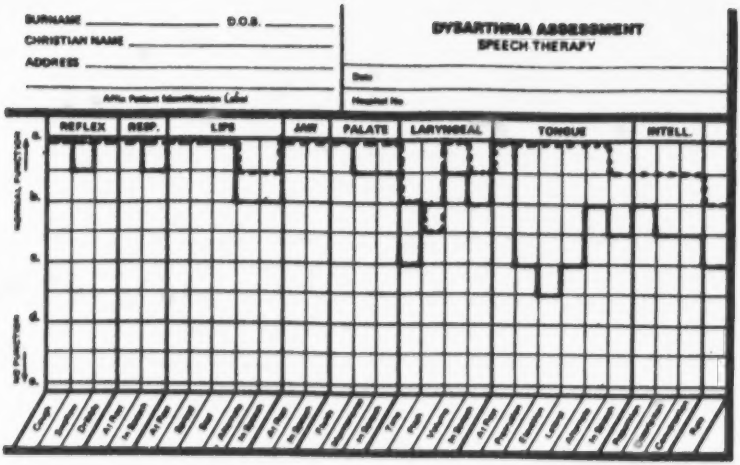

Figure 1. Frenchay Dysarthria Assessment (Enderby 1983) profile of the case at the second (——) and fifth (-----) assessment. Levels a b c d e represent increasing deficiency and have been transformed into numeric scale 0-8 (see table 1).

4. SPEECH AND LANGUAGE FINDINGS

Table 1 shows scores of the Frenchay Dysarthria Assessment (FDA; max = 232), speech rate measured with the reading time (sec) of a short text passage and the average duration of 12 repeated utterances (sec) from the FDA, scores of the Token Test, speech fluency (amount of animals named in one minute), and the aluminium concentration in serum (s-Al; measured during the same month as the speech assessment).

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Table 1. Speech assessment results and serum aluminium concentration during the study (see text).

		Speech rate		Token		s-Al
	FDA	Read.	Repet.	Test	Fluency	(μg/g)
82-10-05	23	39	2.18	35/36	15	0.51
84-04-03	47	55	3.05	34.5/36	16	0.22
84-10-16	27	46	2.24	36/36	--	0.52
85-02-05	20	34	1.98	36/36	20	0.25
85-08-13	14	30	2.02	35.5/36	23	0.24

The results of the Frenchay Dysarthria Assessment indicated a mixture of mild spastic dysarthria and oral apraxia (Fig. 1). The pathological findings were mainly concentrated on tongue movements and laryngeal functions and were most prominent at the second assessment, after which a continuous improvement of functions can be seen. The same trend is evident in speech rate. The intelligibility of speech was rather good all the time. Aphasic symptoms were practically nonexistent; the patient had no naming difficulties, and the Token Test scores were always within normal limits. The patient's reading ability was good and she had no problems in reading comprehension. She had mild dysgraphia which, however, was most likely related to congenital dysgraphia. Speech fluency showed continuous improvement (not measured at the third assessment, unfortunately). An interesting point is that the serum aluminium concentration did not follow the clinical improvement.

No specific neuropsychological disorders could be observed in the assessment by the psychologist and no signs of dementia were apparent. In the following, the most notable disorders in the patient's speech are presented.

4.1. Dysfluency

At the initial stage some sporadic periods of stuttering-like dysfluency and even mutism had been observed after dialysis. The neurologist who first examined the patient reported stuttering, but after that no such features were noticed. However, word- and even syllable-initial sounds were sometimes prolonged, resembling mild forms of stuttering-blocks (Fig. 2). The patient's speech rate was slightly slow, but it

has been improving after the second assessment (Table 1). The dysfluency may have caused the incorrect realization of the phonological quantity oppositions of Finnish vowels and consonants. Long and short quantity degrees of Finnish were not always adequately differentiated.

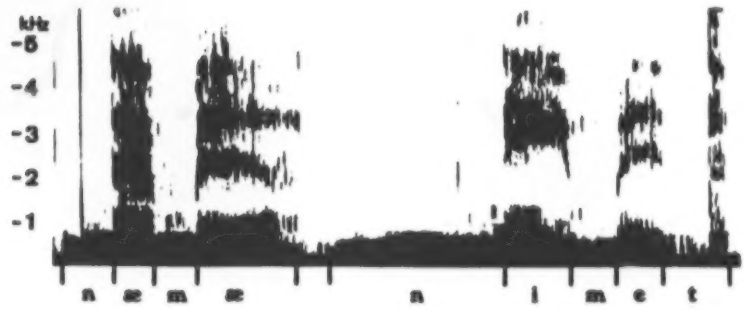

Figure 2. Spectrogram of the words nämä nimet (of the sentence 'näimme vain nämä nimet') repeated by the patient at the third assessment. Note the prolongation of the word-initial [n] of the word nimet.

4.2. Articulation

In general, the patient's speech had a very laboured quality, and some spasticity and difficulties in motor programming of the articulatory movements were obvious. In contrast to the previously described cases, the present patient had almost no trouble with consonant articulation as such. In addition to some sporadic intrusive consonants, such as usva -> usvas (in 'usva nousee suolta') and tutulta -> tuntulta (in 'taulu tuntuu tutulta'), which can be explained as perseveration of syllable structure, the only consonantal error was the tendency of the voiced stops /b/ and /d/, which are not firmly established in the Finnish phoneme system (Karlsson 1982: 57-59), to be irregularly replaced by the corresponding voiceless stops /p/ and /t/. Most of the

difficulties the patient encountered in consonant articulation seemed to be related to sequential organization of complex articulatory movements. In contrast, the phonetic quality of her vowels was often distorted. The distortion did not appear as consistent phoneme substitutions but rather as irregular inability to reach proper vocal tract configurations for vowels, especially for the short cardinal vowels /i/, /a/, and /u/, which showed some tendency to become centralized, so that /i/ and /e/were realized as [e] (or [ɛ]) and /u/ as [ʉ] (or even [y]). The deviating vowel quality may have been an indication of disturbances in coarticulation. A prominent feature in the patient's speech was the inability to execute sequenced articulatory movements correctly and with proper timing. This could be heard, for example, as "wrong" or "unnatural" labial position in consonant articulation in certain contexts, e.g. the labialisation continuing well over the middle /k/ in the word kaukana. These disturbances were most evident at the first two assessments and were later practically nonexistent.

4.3. Voice Quality and Prosody

The patient's voice showed some variability from one assessment to the next. At the first session it was only slightly hoarse. At the second assessment it was worst, with harshness and mild strain-strangle dysphonia. At the next two assessments the voice quality showed clear improvement and was almost normal at the last session. The pitch was slightly monotonous and somewhat lower than normal at all assessments except the last one, when the pitch range was larger. Word stress was not normally distributed (especially at the second and third assessment), phonologically stressed and unstressed syllables tended to receive almost equal stress, perhaps because of the very slow speech rate. This "scanning speech" character disappeared after the third assessment.

5. DISCUSSION

Dialysis encephalopathy is normally - without any treatment - a fatal disease leading to death in about six months from the beginning of the symptoms. The new methods mentioned above - reverse osmosis, which is used to purify the dialysis water of aluminium, and desferrioxamine, which transfers aluminium from tissues to the blood serum, thus making its removal from the body possible by dialysis -

obviously stopped the progression of dialysis encephalopathy in our patient, and even brought about perceptible improvement of functions. No dementia developed and the patient's speech dysfluency never reached the stage of stuttering-like features that are so characteristic of other patients previously described in the literature.

Most of the speech disorders of the present case could be described as "timing problems" in the planning and execution of motor schemata. These timing problems could be observed e.g. as prolongation of word- and syllable-initial sounds, which in its turn contributed to the stuttering-like dysfluency. This impaired temporal coordination of articulatory movements was also seen as deviant coarticulatory patterns and as the incorrect realization of the phonological quantity oppositions of Finnish. The patient's speech rate was slow, perhaps partly in order to compensate the difficulties in executing rapid articulatory movements. This slowing down may have caused the abnormal stress patterns by making it impossible to maintain normal coherent stress-groups.

Besides the timing problems the most notable features of the patient's speech disturbances were the distortion of vocal tract configuration in vowel production - a feature not mentioned in earlier reports - and the hoarseness of voice. Nonverbal motor performance was poor in tongue movements and laryngeal functions.

In general, the patient's speech can be characterized as a mixture of dysarthria and dyspraxia. And we believe that this is, in fact, the case with the patients previously described in the literature, too.

A fluctuating course is typical of dialysis encephalopathy; the condition and performance level of the patient may vary within wide ranges from practically normal to almost total inability to communicate. The fluctuation was not marked in the present case, except for some periods of flu, when the patient's performance was somewhat deteriorated. So we believe that the changes in her performance are not due to periodic random fluctuation of the symptoms, but reflect the improving effect of the reverse osmosis water treatment and desferrioxamine on her brain functions.

5.1. Neural Basis of Communication Disorders in Dialysis Encephalopathy

The communicative disturbances in dialysis encephalopathy include both dysarthria and dyspraxia and progress later to more global cognitive and language deterioration, which suggests that cerebral damage is multifocal and diffuse rather than focal (Madison & al. 1977; Barron & al. 1980). In this respect there is a certain

similarity to acquired dysfluencies/stuttering. Although the neuroanatomic correlates of acquired stuttering are not thoroughly understood at present, most studies have, however, indicated the presence of diffuse or multifocal bilateral lesions in persistent dysfluencies, but some cases of unilateral dominant hemisphere lesions have also been described, most of them transient (Helm & al. 1978; Mazzucchi & al. 1981).

In spite of the symptoms, sometimes very severe, abnormal findings in the nervous tissue have been surprisingly few in dialysis encephalopathy. No anatomical changes have been found at necropsy (Alfrey & al. 1972; Madison & al. 1977; Lederman & Henry 1978; Chui & Damasio 1980). The slight cerebral atrophy and lacunar infarcts in the basal ganglia and thalamus observed by Chokroverty & al. 1976, Baratz and Herzog 1980, and Milne & al. 1983 are not considered to be responsible for the dialysis encephalopathy (Chokroverty & al. 1976). Similarly, the mild CT and NMR abnormalities seen in our case can hardly explain the patient's communication disorders.

The main finding in all previous cases has been the increased content of aluminium in the brain tissue. The concentration of aluminium in the cerebral cortex has been observed to be even 20 times higher than normal control values (Baratz & Herzog 1980). The amount of aluminium in blood serum does not seem to have a positive correlation with the severity of the disorders (Table 1). However, it has been suggested that high aluminium concentration in serum might in fact indicate that the aluminium has been transferred from the nervous tissue to the blood serum (Wills & Savory 1983), thus diminishing the neurotoxic effects of aluminium. This might be the basis of the recovery seen in our patient.

Certain types of EEG changes have proved to be typical of dialysis encephalopathy, i.e. slow-wave activity with bursts of delta waves and high-amplitude spikes (Rosenbek & al. 1975; Madison & al. 1977; Baratz & Herzog 1980). Similar findings were observed also in our patient, the slow-wave activity being most prominent in the left temporal area. The EEG abnormalities have slightly progressed in spite of the clinical improvement of the patient. These changes have been quite mild and they might be due to lowering of vigilance during the recordings, which were made after other examinations on the same day.

The pathogenesis of this disease is not completely understood as yet. The clinical pattern of dialysis encephalopathy is said to reflect more than one etiology, and some other agents may cause similar syndromes (Schreeder & al. 1983). In fact, some sporadic cases of symptoms similar to dialysis encephalopathy have occurred

even in non-dialyzed patients with renal failure (Wills & Savory 1983). This may be caused by the ingestion of phosphate-binding aluminium gels in order to prevent hyperphosphatemia which arises in chronic renal failure (Alfrey 1985). On the other hand, the syndrome does not appear in all patients exposed to high aluminium, and brain aluminium levels may also be high in dialysis patients without encephalopathy (Sweeney & al. 1985). According to Van de Vyver and De Broe (1985) the relationship between the aluminium concentration in a tissue and its toxic effect is not clear at present. Sweeney & al. (1985) have recently suggested that GABA deficiency in frontal, occipital, and cerebellar cortex, as well as in the caudate nucleus and medial dorsal nucleus of the thalamus might explain some features of dialysis encephalopathy. According to them the communicative disorders in dialysis encephalopathy could be due to GABA deficiency in the thalamus.

6. CONCLUSION

Barron & al. (1980) studied 60 patients with chronic hemodialysis in order to find out the early signs of dialysis encephalopathy. They found 21 patients with abnormal speech, 13 of whom had severe disturbances. Since most of these 13 patients were considered clinically normal in medical investigation, it is obvious that a careful assessment of speech and language functions is important for the early detection of dialysis encephalopathy. This will make it possible to start earlier the treatment with desferrioxamine and the dialysate water purification, which have proved effective in dialysis encephalopathy. In future, the scope of investigations into communicative disorders has to be broadened to other metabolic diseases in order to increase our knowledge about neurogenic dysfluencies.

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LANGUAGE IMPAIRMENT IN ALZHEIMER'S DISEASE AND VASCULAR DEMENTIA

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1. INTRODUCTION

The criteria for dementia include a loss of intellectual abilities of sufficient severity to interfere with social and occupational functioning (DSM-III 1980). The clinical picture is dominated by a failure of memory and judgment and there are often also other disturbances of higher cortical functions such as aphasia, apraxia and agnosia. The main types of dementia are Alzheimer's disease (50 % of all patients with dementia) and vascular dementia (30-40 % of the cases) (Sulkava et al. 1985: 1025-1029).

In previous studies there have been made some observations on disturbances in language functions in dementia. However, the characteristics of these disturbances and the differences between the dementia types are not well known.

Obler and Albar (1981: 385-388) maintain that language disturbance is always present in dementia in a more or less severe form. At early stages deficits may be manifest only by careful testing. Later during the course of dementia the symptoms often resemble aphasias of the Wernicke type.

According to several studies anomia is the most common form of aphasia in demented patients, but not the only one. There exist some studies on naming and fluency (Martin and Fedio 1983: 124-141). That the patient produces only a short word list but succeeds in naming tasks with objects is often considered an indication of dementia. Both naming and word fluency are often impaired and there is a high correlation between them. Errors in naming are mostly semantic.

Semantic processes are more likely to be impaired than phonological or syntactic ones. This indicates that semantic functions are dependent on the integrity of higher cognitive functions (Schwartz et al. 1979: 277-306, Bayles 1982: 265-280).

Language tasks that focus on memory functions, such as story retelling and verbal description, seem to be promising for detecting Alzheimer's disease at its early stages (Bayles and Boone 1982: 210-217).

The purpose of the present study was to investigate language impairment in the dementia syndrome and to see if an analysis of language disturbances is helpful in a differential diagnosis between Alzheimer's disease and vascular dementia.

2. METHODS

We examined 21 patients with Alzheimer's disease (11 male, 10 female), mean age 66.2 years (range 53 to 76 years), 25 patients with vascular dementia (13 male, 12 female), mean age 65.0 years (range 48 to 80 years). These patients fulfilled the clinical criteria of DSM-III. Other types of dementia included Pick's disease (2), Huntington's disease (2), Hakola's disease (2), and vitamin B12 deficiency (1). The diagnostic assessment included a neurological examination, detailed medical history, screening laboratory tests, an investigation of the cerebrospinal fluid, a Luria-based neuropsychological test battery, an analysis of language impairment, EEG and CT. Severity of dementia was scored from 0 to 3 according to the tests of orientation, memory and judgment (0 = normal, 3 = very severe dementia).

In the investigation of language we used the Token Test and the Short Aphasia Examination by Maire Lempinen and Sinikka Söderholm. The Short Aphasia Examination consists of seven tests: orientation, receptive abilities, expressive abilities, reading, writing, calculation and motor functions of speech organs. Altogether there are 37 items. The items are selected to test primary language functions and different levels of communication. The results are scored from 0 to 3 in each category. Zero means normal language functions and 3 a very severe impairment.

3. RESULTS

The mean scores for the severity of dementia in the different groups were as follows: Alzheimer's disease 1.85 (range 0.46 to 2.89), vascular dementia 1.45 (range 0.49-2.60) and other types of dementia 1.51 (range 0.50 to 2.96). Thus the degree of dementia in the Alzheimer group was slightly higher than in the other groups.

3.1. Language impairment

Here we report our main findings on language functions in Alzheimer's disease and vascular dementia. The mean scores for language impairment in these groups in receptive speech (rec), expressive speech (exp), dyslexia (dysl), dysgraphia (dysg) and dyscalculia (dysc) are given in the Table.

Table. Language functions in Alzheimer's disease and vascular dementia.

	rec	exp	dysl	dysg	dysc
Alzheimer	1.08	1.28	1.20	1.39	1.53
Vascular	0.64	0.96	1.00	1.20	1.25

All the functions tested were impaired both in Alzheimer's disease and vascular dementia, although the degree of impairment was more severe in all areas in Alzheimer's disease. The language impairment correlated very significantly with the severity of dementia both in Alzheimer's disease ($r = 0.68$, $p < 0.001$) and vascular dementia ($r = 0.78$, $p < 0.001$).

In Alzheimer's disease and vascular dementia with severe cognitive decline there was a severe impairment of all language functions. Patients with severe Alzheimer's disease had difficulties even in the recognition of words and sentences. In early Alzheimer's disease the degree of language impairment was less prominent when compared to the degree of dementia.

Although language impairment could be found also at the early stages of dementia in both groups, there were some differences. The receptive abilities (auditive items, memory, semantics and information) were more impaired in mild Alzheimer's disease than vascular dementia. In expressive abilities word fluency was impaired in both groups from the beginning, narrative speech was more impaired in vascular dementia and repetition of sentences was worse in Alzheimer's disease.

4. CONCLUSIONS

There is a progressive language impairment both in Alzheimer's disease and vascular dementia. It is correlated with the severity of dementia. Different areas of

language functions are affected in early Alzheimer's disease and vascular dementia. In some patients, an analysis of their language might be helpful in a differential diagnosis of dementias. The results of the present study are, however, preliminary. Further studies should evaluate differences in language functions also between normal aging and dementia as well as between pure aphasia and dementia.

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NEUROLINGUISTIC ASPECTS OF FINNISH POSTERIOR APHASIA

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1. INTRODUCTION

The present exposition will deal with some aspects of posterior aphasic narratives. More specifically, we will discuss the repercussions of posterior errors on the lexical structure in a highly synthetic language, viz., in Finnish. (A more extensive analysis of the present speakers' lexical deviations will appear in Niemi et al., forthcoming.)

For the present purposes we will classify the lexical errors rather traditionally as phonological paraphasias and neologisms (see e.g. Butterworth 1979, 1985 for a critique). In that analysis, our major point of interest will be the susceptibility of error location to word and morpheme boundaries. Another main feature of the present exposition will be the fate of the surface case suffixes in posterior narratives.

2. PARAPHASIAS

2.1. Distribution of Lexical Deviations

We will use the distribution patterns of the lexical errors of the two patients as indicates of the linguistic comparability of their outputs (for neuropsychological and neurological data, see Niemi et al., forthcoming). The distributions of the patients' lexical deviations are similar (Table 1). There the phonological paraphasias make up some 60 per cent of the errors, jargon about 20 and the remaining classes, semantic jargon, formal paraphasias, semantic as well as formal cum semantic paraphasias share the remaining 20 per cent of the cases more or less evenly.

Table 1. Distribution of lexical deviations (in per cent)

	Phonological paraphasia	Jargon	Semantic jargon	Formal	Semantic	Formal cum semantic
P1 (N=942)	59	21	6	4	6	4
P2 (N=86)	58	22	4	2	1	13
Is product a lexical item?	NO 80 %		YES 20 %			

The lexicality of the product that has been used here is a tricky question theoretically, since the apparent similarity of the word of the aphasic to an item in the nonpathological lexicon may be an end product of various processes (see Buckingham and Kertesz, 1976) and what is more important still, the homophonicity may be totally unintended by the speaker. Since lexical distortions as such will not be our main concern in the present context we will discard the discussion of their internal subdivision here completely.

2.2. Lexical Structure and Phonological Paraphasias

The lexical difficulties typical of posterior patients usually gather around open, or major class words, such as nouns, verbs and adjectives, while the closed class items, e.g. pronouns and conjunctions, are less severely impaired (see e.g. Marin et al. 1976, O'Connell 1981, Buckingham and Kertesz 1976). This has been taken to imply a partition of the mental lexicon along these lines. (See, however, Ellis et al. 1983 who claim that the frequency of occurrence is a major factor here.) The open vs. closed class difference is also to be seen in our corpus, where about 4 per cent of the running open class words contain a phonological paraphasia, while less than one per cent of the closed class words share the same fate (see Table 2).

Table 2. Phonological paraphasias in open vs. closed class words (per cent running words).

	Open	Closed	Total No. Running Words
P1	4.0	0.6	12027
P2	3.6	0.3	2090

The anomic component characteristic of posterior aphasia is to be observed in the prevalence of phonological errors in nouns vis-à-vis verbs (Table 3).

Table 3. Incidence of phonological paraphasias in nouns and verbs (per cent running words).

	Nouns	Verbs
P1	5.6	2.2
P2	8.7	2.5

2.3. Position of Phonological Paraphasias in Words

Posterior aphasics seem to differ from Broca's speakers in the location of the phonological paraphasias within words. Broca's speakers have more difficulties with the initial segments of words, and in a language like Finnish where the syllable is a significant structural unit, they show a relatively copious number of syllable initial paraphasias. (Niemi et al. 1985.) These difficulties of the anterior patients with initial segments of linguistic units are compatible with the observations that state that they have difficulties in shifting from one act to another (see e.g. Goodglass 1976). The present results show that posterior patients exhibit more errors towards the end of words (Figure 1). Note also that these type of patients also exhibit a similar pattern in the textual aspects of their speech where a discourse may begin in a coherent manner but will often eventually dissolve, perhaps due to failures in lexical retrieval.

A detail that is interesting in Figure 1 where the position of the phonological paraphasias in words is displayed is that the number of errors tends to have a peak

around the 4th to 6th phoneme. Observe that the average length of words in the present data is 6.4 phonemes. Moreover, since we know that most Finnish words carry inflexions that are usually 1 to 3 phonemes in length, it may be that the high fall of the curves in Figure 1 may coincide with the boundary of the stem and suffix(es). That is, we could maintain that phonological paraphasias arise from errors in morpheme selection, rather than from errors in the execution of phonological words. That lexical classes are certainly somehow involved in these paraphasias is shown by the fact that open class words, and within that class, the nouns, are more afflicted by paraphasias than are their complements (closed class items) or quasi complements (verbs).

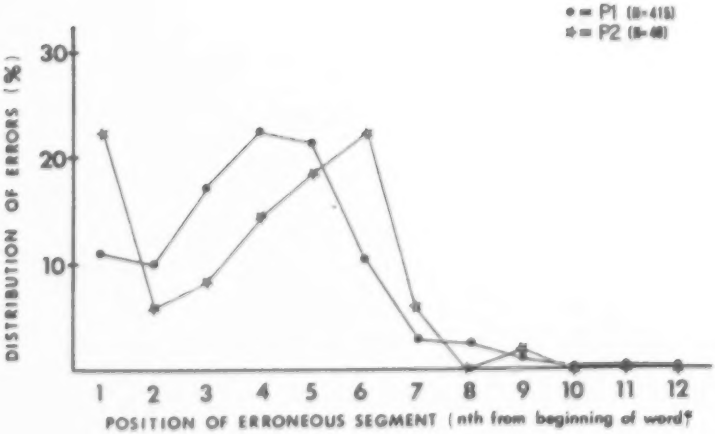
• = P1 (N=415)
✲ = P2 (N=48)
DISTRIBUTION OF ERRORS (%)
30
20
10
1 2 3 4 5 6 7 8 9 10 11 12
POSITION OF ERRONEOUS SEGMENT (nth from beginning of word)

Figure 1. Position of the erroneous segment within the word (in per cent).

In order to dwell into this question more directly we calculated the error rates of the stems and affixes in the present data. We selected the paraphasic words that contained an oblique case marker, a tense marker of verbs and/or a person marker in verbs. Since Speaker 1 (P1) had only a dozen of errors of these kind, we will discuss the larger data of P2. As regards his speech, the ratio of stem to suffix errors is 3.3 (Table 4). And it does not appear to differ from the expected stem to suffix ratio of 3.0. Hence we may tentatively conclude that, unlike true neologisms, phonological paraphasias are not sensitive to intra-word morphemic boundaries. Or, to put it differently still, the paraphasias tend to arise from processes that take place after the word formation rules have applied. However, the susceptibility of the phonological paraphasias to lexical classes (nouns vs. verbs), implies that these deviations do not arise from low level (apraxic) phonological processes, either. The model of language production that could be used as a theoretical point of reference here is an adaptation of Morris Halle's (Halle 1973) design that is discussed in connection with aphasic lexical errors by Buckingham (1981) (see Figure 2).

Table 4. Position of phonological paraphasias in words vis-à-vis morphemes.

	Position or Error (N)		Ratio of Errors	Ratio of
	Stem	Suffix	Stem to Suffix	Stem to Suffix
P1	50	15	3.3	3.0
P2	(7)	(4)	(1.7)	(2.8)

We also agree with Buckingham (op.cit.) when he claims that (pure) neologisms are most probably formed before the application of the word formation rules. Support for this conclusion can be found in the following type of observations (for English, see op.cit., for Finnish, see Niemi et al., forthcoming):

1) Also neologisms are selective as to the lexical class of the item.
2) The affixes, which usually are not formally deviant (see above), are most often inflectional rather than derivational.
3) Neologisms most often obey the morphophonological processes associated with affixation.

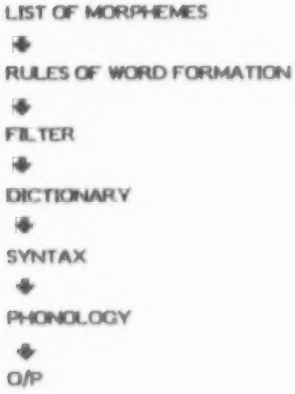

Figure 2. Word-Formation Model of Halle (1973) (adapted from Buckingham 1981).

3. MORPHOSYNTACTIC MARKING

The morphosyntactic marker errors of the present two speakers exhibit two type of observations. For the first, the Finnish cases arrange themselves in the following hierarchy in respect to their sensitivity to substitutions (the data is from Patient 1, who had a large amount of these type of errors, all in all 130 instances, or 7.1. per cent of case markers):

No. of Substitutions (in per cent)	Most								Least
	GEN	ALL	ELA	ILL	ADE	INE	ESS	PAR	NOM

If we analyze the net loss and gain rate for each surface case marker, it will be observed that we will obtain three groups viz. those of, real losers (genitive, allative, elative and illative, perhaps also adessive). The winners will be, as expected on the basis of language acquisition studies (see Toivainen 1980), the nominative and the partitive, while the essive, inessive (and perhaps adessive) will make up the

intermediate group. The percentage values of the net loss and gain analysis are as follows (for details, see Niemi et al. forthcoming):

GEN	ALL	ELA	ILL	ADE	INE	ESS	PAR	NOM
-16	-21	-9	-11	-5	-1	2	11	43

In other words, the almost total agreement between the two case marker "hierarchies" that we have here discussed will suggest that the errors tend to be unidirectional. In other words, a surface case category that is relatively often supplanted by another case suffix will not attract very many errors either. Moreover, the loss and gain hierarchy coincides quite well with the acquisition order of surface case markers in Finnish (see Toivainen, op.cit., esp. his Table 68). Furthermore, since we know that formally, i.e. phonologically and morphophonologically, the markers do not exhibit such striking differences that would explain the hierarchy, it may be that the loss and gain analysis, although it was performed on the surface markers only, also revealed some inherent, cognitive differences between the processing of some cases.

4. CONCLUDING REMARKS

To sum up, we will emphasize the low utility value of the traditional one-component views of phonology and would instead like to claim that the formal aspects of words can be maltreated at various stages of language production. And, as regards the morphosyntax of posterior aphasics, we hope to have shown that surface case marking does carry some consistent and theoretically interesting deviations.

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FRAGILE X SPEECH IN FINNISH: PHONOLOGICAL OBSERVATIONS

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1. INTRODUCTION

The term fragile X syndrome is used to refer to a genetic inherited symptom complex which is linked with a marker X chromosome (fra(X), (q27)). In adult male patients this syndrome is usually connected with severe to moderate mental retardation (Vilkman 1984, Simola 1984).

The fragile X condition is not necessarily linked with any physical anomalies. However, some abnormalities such as large ears, high forehead, high arched palate and EEG changes are among the frequently reported clinical features (Tariverdian & Weck 1982). The earliest description of X-linked mental retardation dates back to the 1940's (Martin & Bell 1943). However, the observation of the marker X chromosome was not made possible until the late 1970's when a specific chromosome culture method (e.g. Sutherland 1979) was taken into routine use. In a chromosome examination the marker X chromosome appears as a constriction of the tip of the long arm of the X chromosome (e.g. Silverman et al. 1983). The balancing effect of the other, and normal, X chromosome has been claimed to account for the fact that female patients are only mildly affected or totally unaffected (Turner et al. 1980).

What makes the linguistic examination of the fragile X syndrome very interesting is the assumption that this syndrome may be linked with a specific language disorder (Lehrke 1974, Netley & Rovet 1982, Bender 1983). In intelligence tests of mentally retarded boys, the scores of verbal tasks have been found to be clearly lower than of those which focus on performance. This trend has been claimed to yield evidence in favour of the specific language disorder (Lehrke 1974). Moreover, and to put it more generally, X-linked abnormalities are interesting because they have been suspected to account for the over-representation of boys in the statistics of mental retardation and delayed language development (Arnold 1970).

The definition of the special linguistic features and disabilities of fragile X patients may yield valuable information as regards the development of therapeutical

and diagnostic practices. It has been claimed that even a limited linguistic dysfunction can stop the general development of the child (e.g. McLaughlin & Kriegsman 1980). In this respect, the prevention of this cessation by means of therapy could be regarded as a goal worth aiming at (cf. Vilkman 1984). The development of the therapy of these patients is very important also when one keeps in mind that the marker X chromosome linked mental retardation may be the second most common syndrome caused by chromosome abnormality in boys (Herbst & Miller 1980). It has been estimated that about 10 new cases are born in Finland annually (Simola 1984).

In the long run, the basic aim in the examination of the speech of fragile X patients will be to define what kind of linguistic problems may lay in the background of relative retardation, or cessation, of the development of a child, and, in addition, to examine what possibilities speech therapy can provide for these problems. The linguistic aims of this kind of study, as will be noticed, are mainly psycholinguistic in nature, and the research practices include approaches such as the phonological process analysis (e.g. Magnusson 1983, Nettelbladt 1984) and the word prototype account (e.g. Leonard 1985, Leonard & Brown 1984, Panagos & Prelock 1982). This paper is a preliminary report on some linguistic features of two physically normal fragile X patients, and only tentative information can be presented as regards the above mentioned general aims.

2. PROCEDURE OF THE PHONOLOGICAL ANALYSIS

The linguistic, i.e., phonological analysis of the data was carried out at the University of Joensuu. Several phonological analyses of various language and speech dysfunctions within a linguistic framework have been performed (e.g. Shrieberg & Kwiatkowski 1982). Thus, the phonological account adopted here may provide a basis for comparison to other types of language dysfunctions, and also to normal language development, described in a similar manner. In order to define the nature of the expressive language disorder, the results will also be (see chapter 4) compared to some linguistic features of Finnish Broca's aphasics (Niemi 1984, Niemi et al. 1985). In addition, we feel that a phonological analysis is desirable, if not necessary, for the development of therapeutical practices, because phonological dysfunctions can in some cases hamper the overall language development (Leiwo 1977). A more

comprehensive discussion of the results obtained from the analysis of these two cases will be presented in Niemi and Vilkman (to appear).

As regards the content of therapy, a phonological process approach may suggest factors which underlie the child's restricted phonological patterns. The analysis might show what processes result in unintelligibility, and, in addition, disclose which processes give rise homonymy, i.e., show what processes underlie the possible basic phonological word types employed by the child. Thus a phonological analysis can also provide a basis for further analysis, e.g. for a word prototype account (cf. Nettelbladt 1983, Leonard 1985, Leonard & Brown 1985).

In the classification of the sounds as correct or erroneous, structurally-oriented criteria were adopted. A speech sound was scored as correct, if it showed allophonic deviations only. The criterion was thus a phonemic one. In other words, a sound was rated as correct, when it could be judged to be an allophone of its target phoneme, i.e., neither an allophone of some other Finnish phoneme, nor that of a distinctly un-Finnish sound (e.g. w, ə, β, θ).

If we had adopted acceptability criteria defined purely at the level of allophones, nearly all of the analyzed speech sounds ought to have been rated as erroneous. In spite of this "gentle approach" of ours, we found hundreds of phonological errors in the speech of our two patients.

3. SUBJECTS AND MATERIALS

The patients discussed here have been studied at the Phoniatric Department of the Central Hospital of Central Finland. The speech samples were recorded by speech therapists Tuula Pulli, Tuula Jalkanen and Armi Mäkelä. The psychological tests were performed by psychologist Marja-Terttu Kuparinen. To the best of our knowledge, our patients are the youngest fragile X patients ever examined.

3.1. Case 1

Case 1 (C1) was born in 1979. He was for the first time examined at the Phoniatric Department at the age of three because of contact and communication problems. An examination was performed to find the marker X chromosome, because his younger brother also showed developmental retardation. The patient learned to walk at 1:2, and the first words were produced approximately at the same

age. From the age of four onwards the patient has attended speech therapy, which has not been without problems because of his hyperkineticity and his inability to concentrate.

C1 was 5;4 at the time of the recordings. His speech sample shows that he can separately produce all Finnish speech sounds. However, his speech exhibits perseveration, clutter, and repetition of initial syllables. Because of these features, spontaneous speech is often unclear and also exhibits phonological and morphological errors. In addition, his speech is often incoherent and wildly associative. Speech-motor evaluation exhibited clumsiness especially with lips. There was also some drivelling. Psychological testing has been particularly problematic because C1 has difficulties in persisting in tasks. Moreover, the patient has clear comprehension problems in verbal tasks.

Performance IQ (Leiter) of C1 was 69 (total IQ and verbal IQ could not be obtained). The patient's intelligence was thus at borderline level. (Note that at the age of three C1 rated 83 in another test which emphasizes performance (M-P).) Physically C1 is in good proportion and his rough and fine motor activities are within normal limits. The patient's hearing is normal, also. The marker X chromosome is found in 20 % of examined cells. C1 goes to kirdergarten and gets speech therapy rehabilitation. Moreover, periods of therapy at the Phoniatric Department are part of his therapy program.

3.2. Case 2

The patient was born in 1976, and for the first time he was taken to the Phoniatric Clinic at the age of 2;2 to be evaluated due to developmental language retardation. The marker X chromosome was found in conjunction with thorough evaluations of retardation. C2 learnt to walk at 1;7 and the development of his rough motor abilities has been within the limits of normal, whereas some clumsiness has been apparent in his fine motor actions. The patient's oral motor skills are poor and drivelling has appeared continuously. Speech therapy was started when C2 was three years old. Hyperactivity, concentration difficulties, low anxiety threshold in tasks and shyness has made the therapy very difficult. In intelligence tests (WPPSI) C2 received a total IQ of 63. Performance IQ was 62 and verbal IQ 56. C2 was thus rated as mildly mentally retarded. The age of the patient was 7;6 at the time of the clinical evaluation. Of these two patients, C2 is clearly the more retarded one.

C2 was 8;0 at the time of the recording of the speech sample. As regards his present speech, typical audible features have included repetition of initial syllables,

fast speech tempo, and clutter, all resulting in difficulties in intelligibility. In addition, C2's speech includes a large amount of perseverations. The patient can produce all Finnish sounds separately and he often uses grammatically correct complex utterances. He produced his first words about the age of two, but two-word utterances were not employed until the age of four.

The patient is physically normal, his ears are within the limits of normal; his face is longish and narrowish. The marker X chromosome was found in 30 % of the cells examined. At the moment, the patient attends a special school for aphasic children.

4. RESULTS AND DISCUSSION

4.1. General

A recently published preliminary report (Paul et al. 1984) on the linguistic proficiency of three 10-13 year-old fragile X patients states that these individuals show a specific verbal dyspractic disability. In addition to mild to moderate mental retardation, the reported similarities between fragile X patients include the following linguistic features: 1) poorer performance on production than on receptive language or nonverbal tasks, 2) poor intelligibility in connected speech despite good performance on isolated words in articulation tests, 3) the use of a very limited number of phonological simplification processes, 4) poor performance in repetition tasks, 5) disfluency, e.g. prolongations and repetitions of sounds and syllables.

As regards our patients, they exhibit language features very similar to those reported in other studies of fragile X speech: they both showed repetition of initial syllables, perseverations and unintelligibility. In addition, C1 exhibited such features as incoherence and comprehension problems in verbal tasks and C2 showed fast speech tempo. These linguistic features are similar to the speech characteristics of children of normal intelligence with developmental apraxia of speech.

4.2. Vowels

To generalize, the pathological phonology of our patients seems to be rather systematic, i.e., failures occur in a systematic manner. As regards vowel errors, the following trends can be mentioned:

1) Back V ==> front V; i.e., there is a clear tendency of fronting; this trend seems to be the strongest one among the vowel errors.

2) Close V ==> ∅; i.e., close vowels tend to be easily omitted. The total amount of vowel errors was 70, 37 of which were omissions.

3) V: ==> V; disappearance of quantity opposition. A rather rare error-type; this is found also in Broca's aphasics. The rareness of this feature can be taken to imply that speakers of a quantity language like Finnish are constrained by the short-long boundary even in the face of severe language impairment (cf. Niemi et al. 1985).

4) Labiality is resistant to error. Also Broca's aphasics seem to exhibit this feature (cf. Niemi et al. op.cit.). This trend is somewhat surprising, because the oral motor skills of both of the patients are rather poor.

4.3. Consonants

In this connection, the most important trends concerning place, manner, and sonority of consonants will be briefly discussed.

As regards place of articulation, dentals seem to be the most sensitive groups for substitutions. The direction of errors of C1 is mainly towards /h/and velars, and in C2 towards /h/, velars, and labials. The basic difference between the two cases seems to be in the amount of labiality errors. Note that the high rate of /h/ can most likely be explained as an error of manner, and not as one of place.

Omissions follow a similar pattern in both cases: dentals are very easily omitted, and velars are the next most frequently omitted group as far as place of articulation is concerned.

As for manner of articulation, the rank order of sensitivity for change is the following: /r/ > SIB > STO > NAS > SPI > /l/ > /j/. /r/, sibilants and stops attract most of the errors, whereas /j/ is always j-like. Laterals, spirants and nasals include relatively few errors.

As regards omissions in relation to manner of articulation, nasals are easily omitted by C1, and stops are not very far behind. C2 is characterized by the omission of stops while other classes show a few errors only.

It seems that substitutions are the most common phonological dysfunction type both in fragile X syndrome and in Broca's aphasia. The proportion of omissions and additions is rather even in aphasia, whereas fragile X patients exhibit only a few additions. (Cf. Niemi et al. 1985.)

Changes in the sonority of consonants were observed to take place along the following continuum:

<------ <-----
STO - FRI - LIQ - NAS - (/j/)
<----------

The arrows above indicate the general trends of change in sonority. It can be seen that stops are usually substituted for fricatives, and fricatives are substituted for liquids and nasals. No sonority changes concerning /j/ could be observed.

In addition, a "loss and gain" analysis was performed as far as both place and manner of articulation are concerned. The results of this analysis are shown in Figures 1 and 2. In these figures the values, i.e., bars above the 0 %-line indicate how often a substitution, a phonological process, took place towards a certain category of place or manner of articulation. For example, the move could be from manner X towards spirants (X ==> SPI). The bars below the zero-line indicate how often the move was away form a certain category, e.g., SPI ==> X.

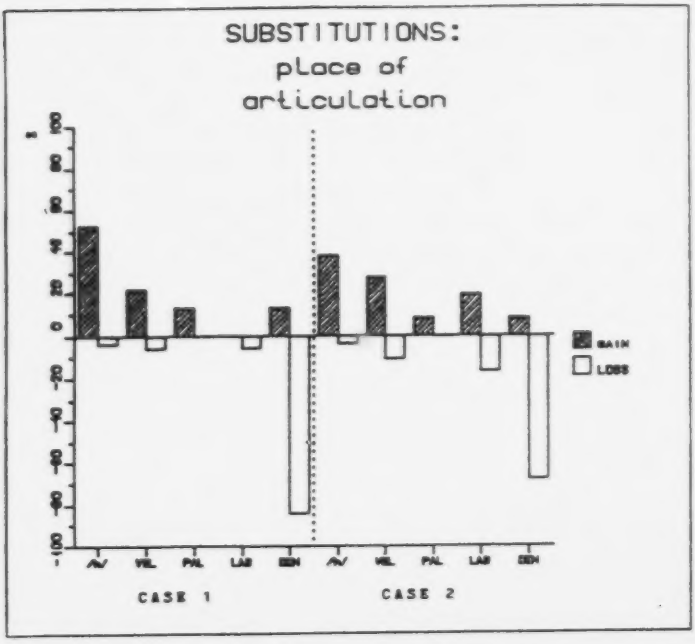

Figure 1.

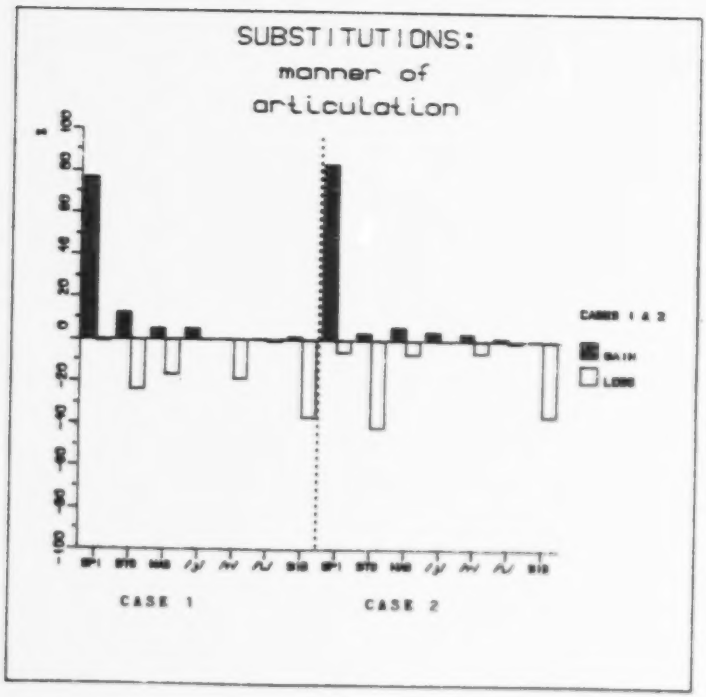

Figure 2.

As for place of articulation, the general trend is clearly rather identical in both cases. The older patient (C2) exhibits perhaps a more stable distribution of substitutions. One could assume that the distribution becomes more and more stable in the course of the development and growth of the patient. The direction of substitutions is mainly towards /h/ in both cases; this is most likely an error of manner of articulation, however. Dentals, on the other hand, seem to be the most unstable category of place with these patients.

Spirantization (h-zation) seems to be the major phonological process concerning manner of articulation. In addition, one can note that sibilants and stops are the most prone categories for substitution with these fragile X boys.

As far as future research, the word prototype analysis may yield useful linguistic insights and, in addition, provide a basis for the development of diagnosis and therapy of fragile X patients. One additional future direction for research might be the comparison of the results obtained here to the linguistic features of Down's syndrome patients.

Acknowledgements

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THE GREAT NEUROLINGUISTICS METHODOLOGY DEBATE

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Currently a major debate rages in the neuropsychology community as to whether the case study is to be preferred over the group study of brain-damaged patients.

The debate first became public at a symposium at the Academy of Aphasia in 1982. Papers from that symposium have been published in several issues of Brain and Language in the following years (e.g. Poeck, 1983; Schwartz, 1984; Caramazza, 1984). Moreover the new journal Cognitive Neuropsychology is devoted to highlighting and exemplifying the benefits of the case study.

Because of provocative statements that not only recommend the case study, but also criticize the group study, practitioners whose primary work has been on group studies have felt obliged to defend themselves. This has led to a healthy evaluation of methodology in neurolinguistics, but the discussion to date has been at the level of advantages and disadvantages of either case studies or group studies. The assumption in such a discussion is that neurolinguists pursue a single goal and at the end of the debate we will know whether the case study or group study will best get us there. I question this assumption.

For a number of years I have been interested in the issue of how research questions develop, and how the techniques and methodology one uses influences both the questions one asks and the answers one gets. Even by framing my questions the way I do, I am contradicting the standard myth about the way research is conducted.

As we all know, there is the myth about science that a question comes to us through inspiration and/or via the muses, and then we sit down to design the best experiment for answering it. Curiously enough, the muses regularly ask questions which appear to be answerable using the techniques we are already familiar with, the equipment we have in our laboratories, and the brain-damaged populations we have some access to. So there is invariably a range of pragmatic factors that determine what research scientists pursue (and what we choose not to pursue), and the techniques and methodologies we use to answer our questions. Moreover, I have a conviction that the methods we use influence and constrain the questions we ask, that is, that the procedure of the myth is in fact often reversed.

In order to gain some perspective in the neurolinguistics debate, I determined first to review the history of case and group studies, as they contributed to the development of neurolinguistics, and then to reflect on the work I have conducted in the field, in order to illuminate my thinking by using the data I know best, that is, the research process I have been involved with.

Historically, of course, neuropsychology evolved as a discipline with a large number of case studies advancing the field. However in a sense it was a group study which triggered the development of the field, namely Broca's observation that over a series of patients with language disorders, it was characteristically the left hemisphere which was damaged and not the right. Such an observation could not have been made, it is clear, on the basis of a case study. Also, already at the end of last century there were group studies of the descriptive sort. For example there was the work of Séglas (1892) on the language of mentally disturbed patients, which included descriptions of the language of dementia. These sorts of group studies were descriptive of the range of phenomena observed, but they were not in the least quantitative like the group studies which were to follow in the 20th century.

With the increasing dominance of behavioral psychology, especially in the United States, group studies became the preferred mode of aphasiological work. Numerical scores could be given for each patient's performance on a task or a set of tasks, and then statistics could be applied in order to test whether there were differences between two groups or two conditions. Naturally these groups were never too large when they involved brain damaged subjects, because no given institution sees great numbers of such patients.

As the field of aphasiology developed aphasia subtypes were identified. Thus it soon became appropriate to study presumably homogeneous groups of patients within a subtype, or compare one subtype with another, rather than simply testing "aphasics". And finding a large group of a given subtype was often pragmatically difficult, so groups sized 6-15 were considered acceptable.

Along with these group studies, fascinating individual cases were often still reported, usually by behavioral neurologists and their colleagues, across the century. By definition what makes a case unusual or interesting is a pattern of dissociation or association which has not been seen before and is not trivially obvious or explainable. Only recently, in the time of the Great Debate has there been a turn to the case study of the individual case that is not necessarily unusually "interesting". Practitioners such as Caramazza who choose the case study method maintain that

virtually any patient with a given neurolinguistic breakdown will prove of interest in extensive testing of hypotheses. Moreover they maintain that the group studies standardly done in aphasia bring together patients who may have a similar diagnostic syndrome, but in fact differ in so many ways that interpretation of combined results from a number of patients hides interesting phenomena and suggests as truths findings which are due to something other than the syndrome.

This question of whether the case study or the group study is to be preferred clearly interacts with my question of how different techniques and methodology interplay with the sorts of questions we ask and the answers we get, a point I will return to below. First however I want to talk about a related set of questions, that of the issue of theory-driven versus descriptive or, as it is sometimes called, pre-theoretical research. Theoretical work generates and elaborates models; descriptive work provides the data from which theories can be constructed, and tested. A major impetus to this question of whether research should be theory driven or whether it should be descriptive, results from the interdisciplinary nature of neurolinguistics.

Indeed, one of the reasons why neurolinguistics is as vital a field as it is, I suspect, is because it results from the convergence of three quite different fields of knowledge: linguistics, behavioral neurology, and psychology. Because the training for each of these fields is substantially different, and the ways in which one acquires knowledge in each of the fields is different, the sorts of questions and the methodologies used by each type of neurolinguist vary substantially.

The behavioral neurologist is trained to see each patient as an individual and to search for the cause of pathology via a listing of the spared and impaired abilities, and to search for the parameters of a patient's history which have proven to be related to the symptoms in question. Clearly this sort of training results in the predilection to appreciate what is special about a given patient, and to do case studies of the sort which demonstrate unusual dissociations or associations of disability.

At the same time, very quickly in the training of the good clinician (I include in this term speech pathologists and neurolinguists doing clinical work and psychologists and neuropsychologists, as well as more direct health care providers such as physicians) something we call "clinical intuition" develops, whereby the clinician can appreciate through some gestalting ability that the patient in front of her or him is similar to another patient seen previously. This appreciation permits the physician to close in on a specific set of questions in working out the logical connections which may rule out or rule in a given diagnosis. Thus the clinician also

has a sense of the individual patient as the member of a group bearing a diagnostic label.

The experimental psychologist, by contrast, is trained to disregard any sorts of "clinical intuition" since they probably result from prejudices and biases. Rather the focus must be on developing hypotheses for truths which will hold across individuals, and thinking through ways to test them. The psychologist does not expect all subjects or patients of a given subtype to perform in a certain way, since human beings are so complex. Rather he or she relies on statistics to determine when trends are significant, i.e., when findings are very unlikely to be due to mere chance. Thus the psychologist is prone to doing large group studies (indeed the groups should be as large as possible) and has, as a rule, great disdain for the case study since any case may be an exception to the general rule.

Note that there is some circularity in the argument that large group studies are necessary in psychology because you can only do statistics on data from large groups. In fact statistics as we know them were developed explicitly in order to conduct large group studies; only today are a new set of statistics being developed for single case studies. Rather than randomizing the selection of the population (which is of course another myth; it never really happens in even the largest group studies), one randomizes the multiple results for analysis.

The linguist, by contrast to both the psychologist and the behavioral neurologist, is trained to observe and infer patterns in corpora of data. Sometimes these patterns are relatively explicit or surface patterns; other times they are "deep" and must be uncovered. For the linguist, truth lies in finding the elegant or parsimonious description of such patterns which accounts for as much of the data as possible, either data at hand or data which may in future be presented. With Chomsky's revolutionary insights of the late 50s ja 60s, linguists have come to believe in a myth of our science, that in linguistics one should be able to choose between two alternative theoretical explanations for a given phenomenon. In fact, however such a procedure is virtually never practiced because linguists seem to prefer to move on to new topics rather than worry old ones. The linguist's approach does not particularly predispose to using either case or group studies. Patterns can be deduced in data from either type of study, though I suspect that the types of patterns which will be evident may differ somewhat, since the case study will demand or suggest more micro-level analyses, while the group study will present more macro-level possibilities.

Within the myth of how psychology is practiced, one works down from a theory. Thus theory-driven work is considered more valuable than "merely" descriptive

work. Linguists too are concerned to present refutable statements, but the form which the refutations may take are not as rigorously delimited as those of psychologists. In psychology, the standard form for research is setting up two alternatives, and then determining a way to choose between them. For the behavioral neurologist whom I have stereotyped as prepared to spot the educational and interesting case, or for the linguist prepared to infer patterns of language behavior in individuals or across groups of individuals and propose theories based on them, the concern for choosing between alternative hypotheses is simply of less interest. Rather the concern is for providing careful descriptions of new phenomena which the theories must eventually explain.

My own work has been primarily with group studies, of larger and smaller groups, but it has also included some case studies, and several of my newer interests, namely the Cross-Language Study of Agrammatism and the Neuropsychology of Talent and Special Abilities, virtually demand the use of the case study. In addition to the cross language study of agrammatism and the Neuropsychology of Talent and Exceptional Abilities, my interests have been in two areas: the neurolinguistics of bilingualism, and the language changes of healthy aging and dementia. Within two of these four fields, bilingualism, and language in aging and dementia, I have been involved in quite a number of studies; the Cross-Language Agrammatism Study by contrast is a single major project involving many colleagues on syntactic production of agrammatic aphasics in 14 languages, and the work on the neuropsychology of talent is in its early stages. There our immediate goal is a co-edited book on the topic to bring together what work there is on talents such as calculating, hyperlexia, chess players, music, art, and learning a foreign language like a native after puberty. The long term goal is a series of case studies to permit us answers to the questions about how the brain is organized for special talents.

Our work in bilingualism and on aging by contrast, has been virtually composed of relatively large scale studies of one or another sort. The groups involved were larger in the cases of healthy bilinguals and healthy elderly, and smaller in the cases of harder to get populations such as the demented patients and the aphasics we contrasted with them. The Cross-Language Agrammatism project is a curious combination of the case study concept and the group study concept as I will elaborate below.

My methodology for thinking through this paper was as follows: I simply reviewed the research I had done, in each instance asking what the research question

had been, what type of study it had been - initially I simply asked whether it was group or case study -and what the results or answer had been. Of course at the time that we designed the studies, it often seemed that our methodology was the only appropriate one to use. In thinking for this paper, however, I pushed myself to ask if we had changed certain parameters of the methodology would we have had to reshape the questions and would we have obtained different results?

As to the type of study, although I initially simply intended to determine whether it was a group or case study, I soon found myself differentiating between large groups and smaller groups, and between different types of case study. I also considered the order in which various components of the scientific method had in fact been carried out, e.g., whether the data were collected after the question had been posed, or whether the question was posed on the basis of data already collected by someone else. I suspected it had also made a difference whether the tests were designed to answer the specific question, or whether previously used tests of other investigators or of our own were employed to give the answer. One question I began to ask was whether the source of the research question was from clinical experience or from experimental experience, whether one of our studies followed up another one in linear fashion or whether it developed to fill a gap in order to be comprehensive.

I won't be able to review all my research in this paper, but I have selected examples of sufficiently different techniques and methodologies to justify my points.

The largest group studies I've been involved with have been the studies of healthy language and aging. The basic question which motivated Martin Albert, my collaborator, and myself was: Are there changes of language with healthy aging? Predicated on the assumption that there would prove to be subtle changes, we asked: If there are changes, what is their nature? Thus initially our research called for pure description. And we set up a major research protocol to look at all aspects of language: laterality, naming, comprehension, automatic speech, discourse, and metalinguistic abilities. Our first set of papers described the quantitative and qualitative changes we saw in some language abilities - naming, comprehension and discourse in particular - and the lack of changes we saw in others - laterality and automatic speech, for example.

In the next phase of this project, as we have a better sense of what changes there are and which do not occur, we have begun to ask what the changes relate to. For example, do they relate to the individual's history of alcohol consumption, or of

bilingualism, or do they relate to gender differences across the lifespan? Do they relate to performance on other language tasks, or does aging affect different language tasks independently? And do changes in language performance in the elderly relate to non-language neuropsychological abilities such as memory and attention?

In the third phase of this project we are developing theoretical hypotheses to test, so that we can use our data on naming in aging, for example, to learn more about the naming process and the processes of cognitive change related to age.

For the extensive set of projects we undertook we recognized that we would need large numbers of subjects in order to see the relatively subtle changes we expected. We assumed that because language abilities are so diverse, and aging so individualized not all elderly would show them. Thus we would need to look for means, and work with statistical differences rather than the striking dissociations one may see as the result of frank brain-damage. Our statistician told us we needed to have a sixteen person cell for each gender, in each of the four decades we planned to test, the 30 year olds, the 50 year olds, the 60 year olds, and the 70 year olds. Although many before us had done studies of language in aging by comparing one younger adult group with one older adult group, we assumed that it would be valuable to get four data points rather than two in order to see whether there was a progression of changes with ages or a drop-off, presuming changes were found. Our initial assumption proved true that there would be substantial intra-individual variation in language performance, which would mask patterns of change across the larger group. Indeed, as in many of cognitive studies of aging, our data evidenced an increase in standard deviations with increasing age.

I will not be able to go into detail on our findings here. In brief summary, however, I will report that in certain of our neurolinguistic and linguistic realms we found no differences with age, such as the tests of laterality and automatic speech; on other tasks such as naming (and this was equally true of common nouns, proper nouns, and verbs) performance declined with some qualitative changes in response type. For comprehension, by contrast, while correctness scores declined, there were no qualitative differences seen despite the fact that we were explicitly looking for them in those tests. With discourse production, we saw an increasing elaborateness with age which was linked to better performance. In order to make such summary statements, of course, I ignore the individual differences and the range of variation. So, using such a methodology I am able to answer the question of whether there exist subtle changes for population groups, I am unable to predict what is happening

for the individual. There is a certain irony in the realization that one cannot generalize from a case study but nor can one generalize to all elderly as individuals from a group study.

Our series of studies on language changes of dementia provide a certain contrast. Originally we had thought we would give the demented patients the same tests we were giving the healthy aging patients. However it soon turned out that the demented patients were performing for the most part so much worse than the healthy aging, that only those tasks which were too easy for the healthy elderly could we use with the demented patients, such as the tests of automatic speech, in which we ask subjects to recite the months of the year or the numbers from 1 to 21. Most of the tests which were interesting enough to provide a range of performance among the healthy subjects would have been unkind to give to the Alzheimer's patients since they either could not have caught on (as in the case of watching a television and pressing a button to demonstrate comprehension) or they would have failed severly (as in the case of the 85 item naming task).

Instead, as I spent more time with demented patients interviewing them, a different set of questions were motivated for clinical purposes.

For example since the literature on language changes of dementia was so sparse when we started, it was important to resolve the apparent contradiction that people reported both logorrhea and muteness in dementia. Over the course of working with patients in different stages of the disease, it became clear to me that logorrhea was associated with a middle stage of the disease, and muteness with an end stage. Indeed I was able to discern a progression of six stages from early to late, which we describe in greater detail in Martin Albert's book The Clinical Neurology of Aging (1984). Interestingly, two of the stages were particularly compelling to the linguist/aphasiologist in me; the early-to-mid stage when the patient looks something like an anomic aphasic, and the mid-to-late stage when the patient looks substantially like a Wernicke's aphasic. This observation led to two additional studies, one to distinguish the empty speech of the Alzheimer's patient from that of the Wernicke's aphasic and the anomic, and the other to test naming in these patients and relate it to that of anomics and normals. Again, this question of how to differentiate the speech production of the Alzheimer's patient from that of the aphasic had a certain diagnostic impetus because I was regularly asked by neurologists to judge whether a given patient was in the relatively early stages of the dementing disease or whether he or she was aphasic from causes other than dementia. I selected a set of language tests to give demented patients over time,

and I developed a certain clinical intuition which allowed me to evaluate a patient alongside my memory for patients seen previously. The study of empty speech permitted us to quantify impressions (Nicholas et al, 1985). At first I was distressed that out of the 14 items we chose to look at: (including empty phrases, deictic terms, anaphora without antecedents, indefinites, paraphasias, neologisms, and lack of discourse conjunctions) only three differentiated the demented from the Wernicke's aphasics patients. But then I realized that this lined up with everyone's previous clinical sense that the empty speech was really quite similar across the two groups. The differences, by the way, lay in the fact that the Wernicke's patients produced neologisms and verbal paraphasias significantly more frequently than the Alzheimer's patients, whereas the Alzheimer's patients used a broader range of conjunctions, particularly the logical conjunctions such as because and although, as compared to the Wernicke's patients. Had we compared more extended discourse from a single patient with Alzheimer's disease with that from a Wernicke's aphasic, we well might have obtained the same results, although it is also possible that certain of the particular phenomena observed to distinguish the groups might not have obtained with sufficient frequency to permit statistical differentiation.

My clinical work with demented patients suggested a further study. I was impressed with the fact that the comprehension of the mid and mid-to-late stage patients was quite poor, for reasons which appeared to me to be attentional. In the Wernicke's patient, by contrast it is a language area of the brain which is destroyed. It made sense to suggest that reading materials be given the Alzheimer's patient so that there were two modes of input, so memory would be less called upon, and attention would be reinforced. This study was actually conducted as a relatively small group study, with 9 patients each getting the same battery of comprehension materials from the Boston Diagnostic Aphasia Exam in each of three conditions. The choice of a small as compared to a large group here, as in many other instances was pragmatic rather than theoretical; a student was doing it as her honors thesis. However the results were encouraging with seven out of nine patients performing better in one of the written modalities, and the differences reaching significance for the most complex subtests of comprehension. In a case study we might have tested one of the two cases who were not helped by written input, and might thus falsely have concluded that it would not help other patients with Alzheimer's disease.

By contrast to the work on healthy aging, my work in bilingualism regularly involves somewhat smaller groups of 10-15. Interestingly, they would be considered rather large groups if they were studies of brain-damaged subjects. The work on

laterality in bilingualism presumed that two groups of 24 bilingual and monolingual subjects would be representative of their respective populations. The one bilingualism study which appeared to be based on a larger group, was the review of bilingual aphasia cases in the literature available to us at the time (Albert and Obler, 1978). In that study we looked at 106 cases which had been published, in order to ask the question which had dominated the field for the last century as to which language comes back first in those polyglot aphasics who show differential recovery - that is a difference in aphasia between the two languages which could not be predicted on the basis of the patient's knowledge of the two languages premorbidly. Was it the rule of Ribot that the first learned language returns first, or the rule of Pitres that the language used at the time of the accident returns first, or some other factor such as handedness, or the specific language spoken, or age of acquisition, or age at onset of aphasia? Thus we coded for all the personal history, aphasia type, language history and etiology variables available in order to see whether there was anything else which predicted which language would return first. As it turned out we did get significant results in favor of the rule of Pitres, whereas the rule of Ribot held with merely chance frequency. But we could say nothing statistical about most of the other factors we had been interested in, since the data were too skimpy on any given parameter (Obler and Albert, 1977). With this study, I first learned that what appears to be a large group is not necessarily so if one does not personally control the collection of raw data. Thus we had handedness data for something like only 15 of the subjects, and in many cases the language testing was skimpy, and in only a few of the patient's languages. In a few cases even the gender of the subject was not given. As a result we were unable to find any of the other possible correlations which might have proved of interest.

Indeed the case studies we have done on polyglot aphasics have proved at least as informative. Of course from the case studies one cannot answer questions about how the group as a whole responds, but one can instead find a dissociation, such as that reported in Albert and Obler, 1978, with different aphasia types in each of two languages. Because this is such a rare phenomenon, we were fortunate to locate and test this case. Note that this sort of dissociation can hardly be looked for in a large group study, and yet it is very important for documenting the range of ways in which language can be organized in the brain of the bilingual.

Certainly the largest group study I was involved with was a study of handedness in Israel (Silverbert et al, 1979). As I was coming into the field of neurolinguistics, like many I became particularly aware of how many left handers there were in the

world. Probably due to the circumstance of my being in Israel I soon hypothesized that Israelis as a group had a greater proportion of left handers than I recalled from my limited upbringing as a right hander in the United States. Such an epidemiological study has to be done on a very large scale in order to have data to compare with other studies in the field, so we tested 2,000 subjects, bringing questionnaires from classroom to classroom. In the end we discovered that there are virtually no handedness differences between Israeli Jews and the rest of the world. The lesson this taught me is that group studies do not necessarily provide momentous discoveries proportional to the largeness of the group!

Because of an age-related finding in the study of Pitres, namely that while the group as a whole followed the rule of Pitres the subjects over age 65 did not, Marty Albert and I decided to consider the relation between aging and aphasia type which had not been studied previously (Obler, Albert, Goodglass and Benson, 1978). This research was conducted on a fairly sizable population drawn from about 600 patients seen by Frank Benson at the Aphasia Research Center. We narrowed our sample to a fairly homogeneous set, selecting all 167 patients who had aphasia resulting from stroke in right handed white males who fell into clear cut diagnostic groups of aphasia, and discovered that the Broca's aphasics were significantly younger than the average and the Wernicke's aphasics significantly older. Although this finding has been replicated in at least 11 aphasia research centers aroung the world, and demonstrated by Miceli et al. to be true for patients with tumors as well, the explanations for the findings are elusive. The interesting explanation of course would be that the brain substrate for language reorganizes itself between the early 50's and the mid 60's. Less interesting explanations would be that humans tend to get different sorts of strokes and tumors at different ages.

In a follow-up study with J.P. Mohr and Lou Caplan (1981) we employed the Harvard Stroke Registry, another seemingly large scale data set, in order to look for correlations between etiology, lesion location, and age. As in the previous study, the fluent aphasics were significantly older than the non-fluent aphasics. The strongest finding for the interesting explanation, that the substrate for language reorganizes across the lifespan, would have been if 60 year old patients with anterior lesions had posterior aphasias. But we did not see such a correlation. On the other hand none of the other correlations held true either. For example, there was no significant finding of increased numbers of posterior strokes with increasing age. We are forced to conclude then that the initial observation that older aphasics are more likely to evidence Wernicke's aphasia still holds true, but our attempts at large group explanation have not succeeded.

An alternative approach I fantasize, assuming the appropriate volunteer or volunteers could be found, would be to do a case study or a small group study using a pet scan or the cortical stimulation technique longitudinally. Once each year between the ages of 50 and 70 the patient would be tested in order to determine the extent of his or her language area of the left hemisphere, and within the language area, the extent to which anterior and posterior areas were responsible for comprehension and syntactic production. If the interesting explanation is correct and true for all people, it should be possible to demonstrate that it is true for one individual on the basis of a case study. Given our current set of beliefs that the brain substrate for language is fairly fixed after puberty, even a single case would be so stunning as to force us to rethink our set of beliefs about the development of brain substrate for language. But again, to the extent that we wanted to generalize beyond what might possibly be an individual freak case, we would need to do larger scale studies.

The clearest instance in which I needed to do a case study was in learning about the neuropsychology of talent and special abilities. My interest in this topic was piqued when I heard Dorothy Aram talk on hyperlexia, and realized that to study it or indeed to study any talent would be to reverse the standard neuropsychological paradigm of looking at deficits against a background of normal abilities. In studying the neuropsychology of talent one focusses on the talented ability and tries to determine its components, as they relate to the background of merely normal abilities, or in the case of idiots savants, as they relate to poorer than normal abilities. We have attempted two sets of case studies in Boston, one on exceptional second language learners and the other on natural speed readers. The study of natural speed readers has been delayed because they are extremely hard to find if indeed they exist. We do believe we have located at least two. But for one, her overall intelligence is so high we would not be able to find any of the dissociations we would prefer to find, and the other lives at a great distance - another pragmatic constraint. Two others who volunteered demonstrate very fast reading but with poor enough comprehension that they are not what we meant when we said we wanted to study natural speed readers!

With the exceptional second language learner, by contrast, we have been able to locate several in the Boston area and have completed testing on one and almost completed it on the other two. While it might be of interest to study large groups of such subjects, they are simply not available, and in any event intensive work is

required to look for dissociations between different neuropsychological abilities. In the study of the first subject reported by Novoa et al., we found an interesting dissociation between CJ's overall memory abilities and his memory for language. When given a word list of 20 items, for example, he remembered no more than the normal person upon immediate testing. However when tested two weeks later, he remembered a majority of the words, when the normal can remember only a few. We also found a dissociation between his overall IQ, which was merely normal, and his language learning abilities. The third dissociation we found was among the subtests of the Modern Language Aptitude Battery (Carroll and Sapon, 1959). This test of John Carroll's was devised to test the various skills which go into good language learning ability. Our subject CJ did well on all of them except the ability to abstract grammatical rules. Somehow his language learning abilities permit him to learn language without this ability to abstract rules. Of course we cannot conclude that the next subject we test will not be good in this ability; indeed the fact that this subtest remains in the test battery after Carroll took out all the redundant tests argues that there are subjects for whom that skill is valuable in good second language learning. What we can say is that the ability to abstract grammatical relations is not crucially necessary for skillful post-pubertal second language learning ability.

In addition in that study we found an interesting qualitative result whereby CJ appears to prefer form over function. On the similarity subtest of the Weschler Adult Intelligence Scale, for example, when asked what work and play have in common, CJ reported that they both have four letters, rather than that they are both human activities. When asked what a statue and a poem have in common, he cleverly replied that they both have lines. This focus on linguistic form does not get you many points on an IQ test, of course, but we suspect that it is one of the components which contributes to CJ's superb second language learning skill.

Had we studied a number of exceptional language learners in a group study, many of the specific dissociations we found in CJ might have washed out. After all, we do not claim that all exceptional second language learners have exactly these abilities, but rather that this constellation of abilities makes it possible for CJ, an otherwise normal human being, to be an exceptional second language learner.

Let me turn finally to two examples of hybrids between case and group studies. The first is a study of pragmatic abilities in dementia which developed out of my changing focus of interest within dementia. It is a good example of the sort of small case-group study that some advocates for the case study approach consider ideal. In

essence, each subject or patient from a small group of very homogenous patients is studied extensively. This permits one to see patterns across the patients because of their homogeneity, but also to exclude as non-universals those points on which patients differ. This study evolved as we realized that no one had considered the pragmatic abilities of end stage dementia, which were reported by Irigaray (1973) and others to remain until quite late in the disease. Around the time I realized it was worth wondering about this, a student came along who was looking for a master's thesis project, and she found this study interesting. The methodology we employed was to do intensive analysis of the pragmatic communication behaviors of as many subjects as possible, in order to document the range of remaining abilities, and to determine when they are used appropriately and when inappropriately. Such a study which sets out to describe the range of behaviors is clearly an early study in a field which has been untouched. Irigaray's 1973 study of language in dementia took a similar tack. No numbers are generated at this early stage, but rather the boundaries of phenomena to be studied are staked out. This could actually have been done on a single case study individually, but one would have no idea how representative it was until a group of cases with presumambly the same diagnosis were studied. And our clinical intuition told us that there would be differences in the sorts of communication behaviors which patients retain into the late stages of Alzheimer's Dementia.

In fact, the way we are carrying out this study is a combination of the case and group study in that each case will first be studied and analyzed individually. One of the difficulties with Irigaray's ground breaking larger scale study (she had 53 subjects total) was that it treated each language ability in a separate chapter. However one could have no idea how two or more language abilities clustered for a given patient. Our strategy in this project of looking at the pragmatic abilities through extensive work on each of 9 patients in order to talk about clustering of abilities within each patient. Then we will compare across patients to see whether there is a given hierarchy of preservation, a given cluster of pragmatic abilities which is regularly spared, or whether there appears instead to be substantial individual differences and dissociations.

The second hybrid between a case study and a group study is less orthodox in methodology. It is the Cross-Language Agrammatism Study which Lise Menn and I are coordinating and which involves my host, Jussi Niemi, and several others in attendance at this meeting. The question we ask in this work is whether the language structures of different languages interact differently with agrammatism.

Agrammatism, as you know, is that particular form of aphasia in which the patient omits word endings and some functor words; the resulting speech sounds more or less telegrammatic depending on the degree of severity of the agrammatism. Actually theoretically it need not be word endings which are dropped, but the century of study on agrammatism has been largely done in Indo-European languages where word endings mark the inflections; in a language such as Swahili with productive syntactic use of prepositions, as Traill (1972) reports, these also can be impaired in agrammatism.

In order to answer our questions, one could, I suppose, do a study of a bilingual or, better, a multilingual aphasic who had agrammatism in all languages. Ideally the patient would have been fully fluent in all the interesting languages of the world; Chinese, Japanese, Finnish or Turkish; an American Indian language, and several African and Melanesian languages. Waiting for such a patient to turn up proves prohibitive. And to make it worse, only 2 % of all aphasics, I have read, are agrammatics. So, we determined instead to work with colleagues who could locate agrammatics in different languages, and to ask each to do two case studies, to compare them to each other so we could see what individual differences could be ruled out as not due to differences in the language structure, and then compare the agrammatic cases to normal controls in order to see what language errors were more normal slips of the tongue. The result will be our large sourcebook, currently titled Agrammatic Aphasia: Cross-Language Narrative Sourcebook which will permit us all to do group studies by looking at differences across patients.

Here we have a substantial number of questions which can be answered, looking both at universals and at differences especially between languages. For example we have asked our participants to write about the special features which their agrammatics show in their language; if both agrammatics show a given pattern, we are more likely to assume that it is the language structure in conjunction with agrammatism which brings it about; if only one patient shows the pattern, we ask whether that patient is the more impaired, or whether there are different subtypes of agrammatism in any language, a conclusion we are coming to at this point. We also ask which features are regularly preserved that one would have expected be omitted in agrammatics, given the standard definition involving loss of inflectional endings and functors. Indeed we ask our participants to compose a hierarchy of deletability of morphemes when this is possible.

When there are differences between the two agrammatics, of course, we cannot assume they relate to the language or the degree of the disability; we first ask our

participants to rule out dialect and education differences and potentially handedness and gender differences. One technique we borrowed from linguistics is to ask what sentence types occur. A finding we have across many languages particularly evident in Finnish, for example, is that the agrammatic patients actually only attempt a limited set of syntactic types. This finding would not have been noticed if one simply performed error analysis, or if one only studied agrammatic patients without having normal controls against which to measure their performance.

Another question we will be able to address by this hybrid case and group study is the dissociability between different language modalities such as reading and speaking and between comprehension and production. Thus we already know that for some of our patients there is a good correlation between agrammatism in reading aloud and speaking, and for others there is no correlation whatsoever. Likewise with respect to comprehension, as has been posited on the basis of certain cases and indeed small group studies, there are patients in our study for whom comprehension of syntax is impaired in ways similar to their production. However there are also patients for whom comprehension is simply not impaired, so we must conclude that comprehension disturbance is not universal in agrammatism, and thus that the syntactic processor for comprehension must be dissociable from the syntactic processor for production at least for some humans.

Finally we ask our participants to address the several explanations of agrammatism which have been proposed over the years in order to see whether we will see different ones supported on the basis of different languages. As you may know there had been functional explanations of agrammatism and phonological explanations as well as morphological explanations and more processing explanations such as ones based on words' position in the sentence and sentence length. Clearly such a question is answerable as we have set up the study, but I do not have the answer to give you today.

Obviously had we done only a case study in each language, we would already be restricted in our conclusions beyond what we can say by testing a pair of patients and a pair of controls. On the other hand had we done a large group study, certain of the dissociations we see in an individual patient and between patients might have been masked due to averaging. So in the end I conclude that this combination of extensive testing along the lines of the case study and attention to the clustering of behaviors in the individual has worked well in this study which also requires relatively small group analysis.

One conclusion I was surprised to come to from my review of the research I had been involved in was that there was a wide range between the pure case study and

the ideal large group study. For different research projects "large" in terms of group meant a substantially larger number than for other sorts of subjects. Clearly there are pragmatic reasons involved such as the amount of time the investigator will have, and the likelihood of finding a number of a given sort of patient. However I now believe, contrary to the cynicism with which I started working on this paper, that it is not pragmatic variables alone which determine the size of a sample. Rather on top of the pragmatic variables which say that it is harder to find brain-damaged patients than healthy patients, and harder to find specific interesting subcategories of brain-damaged patients than it is to find just any brain damaged patient, there are also compelling scientific reasons to take unusual cases more seriously, to give them greater weight, than cases we believe to be more run-of-the-mill or normal.

Moreover, I realized, the magnitude of the data to be analyzed is often virtually the same in a large study, a small group study, a case study, or some combination of them. When one has a single case, one generates substantially more comprehensive data than when one is simply asking 10 handedness questions from a sample of two thousand. This phenomenon is being formalized and addressed with the development of single case study design and statistics today. The debate which polarizes discussion around case versus group studies, then, is inappropriate on two grounds, first, because there is a viable continuum between the two poles, with variation between the two ends of the continuum, and second because the amount of data to be analyzed can be surprisingly similar.

What the case study buys one, in essence, is the possibility of demonstrating a clear-cut dissociation, and the possibility of claiming that we see one example of how a human's brain operates, and how language information is processed in at least one person. Also, a case study can contradict any generalized assumption that "All human beings X". What the small group or the large group study gives us is population trends, the possibility of predicting the likelihood that the next patient or subject who appears to fall into a group will share those additional characteristics that you have discovered via the case study. It is possible to reveal small but systematic changes in a population that might not have proven significant in any individual case study. Moreover it is important to note that only in doing group studies can one find what we call individual differences, i.e., differences between subgroups of a population; with the single case study, one cannot make claims for group differences. Of course one can do group studies and not look for individual differences or sub-group differences; but if one plans to look, they can only be found

in the group studies. These constraints on the sorts of findings one can have are essentially the constraints on the sorts of questions one asks. So I conclude that the myth is certainly not fully true, that we ask the questions and then go out and choose the appropriate methodology. Nor is the converse the truth, that the methodology we use fully determines the questions we may ask. Rather there is an interplay between the techniques and methodologies we use and the questions which they predispose us to be interested in, and therefore the answers we will get. At the same time there is an interplay between the questions which come to us based on our previous research or our readings in the field, or insights from the muses even, and the ways they direct us to modify a given methodology so it permits us to approach an answer to the question we have.

There is a certain logical progression to the sorts of studies the behavioral scientist may do. First, in the early stages of approaching a new topic, we need a phenomenological study to describe what may be considered pertinent to the topic or questions. This can just as easily be a case study or a group study. Then we need a study to look at the frequency of occurrence of the phenomenon and its elements and perhaps how they interact. Study of different subgroups can follow. Clearly this requires more than a single case design. On the basis of results from such studies one then designs group studies to see how universal a phenomenon or a cluster of phenomena is. These can be descriptive studies, or they can be theory-driven on the basis of hypotheses.

Writing this paper has made me feel better about the term "pre-theoretical", which in some neurolinguistic circles is said in a derisive tone about work which is not strictly theory-driven. For me, it is the pre-theoretical ends of the field which are the interesting ones to work in, and I now realize that this can be done through case studies in the early pre-theory, and through group studies in later pre-theory. By the time the work gets to be "theory-driven", it has always seemed to me to be too constrained, or "theory-limited" to be of interest any longer. But I hasten to say that that is a reflection of my personal intellectual style and taste; I would hardly prevent a student from pursuing a theory-driven question, or even think the worse of a fellow researcher who preferred to work in that mode.

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CLINICAL LOGOPEDICS: A DIAGNOSTICAL REVIEW OF SPEECH AND LANGUAGE DISORDERS IN A GROUP OF HIGH-RISK CHILDREN

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1. INTRODUCTION

It is evident that slow development of neural processing and neuro-musculator immaturity are factors in the disorders of children's speech and language development. The syndromes of the speech disorders of children are in close connection to the immature functioning of the central nervous system. Disorders can also be consequences of the delayed general development. Among the children of five years of age a wide spectrum of different delays in communication ability can be found. Most frequently this originates from the individual developmental processes in the earliest stages of life which are strongly influenced by the environmental circumstances. The early histories of some children show risk factors in the neonatal period. The influence of these early risk symptoms even as late as at the age of five has been shown in many studies (Calame & al., 1976; Dahlby, 1970; Ehrlich & al., 1973; Saarnivaara & al., 1976, Michelsson & al., 1981). It is common that the abundant existence of both articulation and language disorders at the age of five call for the clinical logopedic examinations. The careful anamnesis of etiological factors at the neonatal period facilitates speech diagnostics in clinical logopedics, as well as planning the amount and type of speech therapy. Language disorders form a very heterogenous group as to their etiology, non-linguistic symptoms and handicaps, their severity and linguistic characteristics (Nettelbladt, 1983).

Our objective here is to clarify what kind of correlations can be found in the speech behaviour of high-risk children in comparison with children without neonatal risk complications. For this purpose observations based on clinical logopedics are collected and on the basis of them the status of phonological development is compared to neurological maturity as one aspect, and psycholinguistic performance to the existence of phonological disorders as another aspect. In addition the mutual

correlations of these aspects are assessed. We have particularly concentrated our work on the articulatory ability of the five-year-old risk children (N = 430), and we have applied a cross-disciplinary diagnosis (child neurology, speech pathology, psychology) in order to find out to what extent certain risk factors have influenced speech behavior, language and psycholinguistic abilities.

The results are compared with the results of the control group (N = 115) consisting of children of corresponding ages, who had not suffered of any risk factors during pregnancy, delivery or the neonatal period (Michelsson, 1981).

2. MATERIAL

The children in this sample were born at the Institute on Midwifery in Helsinki in 1971-1972 and in the newborn period they had one or more of the neonatal risk factors (Michelsson & al., 1981). When the children were five years of age the cross-disciplinary investigations were carried out at the Department of Pediatrics in the Helsinki University Hospital 1976-1977. Altogether 856 five-year-old risk children were examined by pediatric neurologists, speech therapists and psychologists during the years 1976-1979.

A control group was investigated with the same test methods at the age of five.

3. METHODS

Table 1. TEST METHODS OF THE TEAM (1976-1979)

- neurological screening (Bax & Whitmore)
- vision (Sheridan)
- hearing (audiometer)
- psycholinguistic test (ITPA) stadardized in Finland
- logopedical screening
- visual perception (Frostig)
- draw-a-person (Goodenough)
- parent's questionnaire
- teacher's questionnaire

The neurological examinations comprised the neurodevelopmental screening test for five-year-old children developed by Bax and Whitmore (1974). Its Finnish version KAM has been standardized by Michelsson, Ylinen, Donner (1982).

Logopedical screening examinations were based on the methods of certain specific areas of clinical logopedics (Table 2).

Table 2. LOGOPEDICAL TESTS

- articulation test
- speech motor performance
- language behavior
- rhythmic coordination in visuomotor performance
- concentration ability
- observations of rhythm, voice and nasality
- ITPA, Illinois Test of Psycholinguistic Abilities (Finnish test version), auditive subtests

The phonological tests comprised an examination of sound-articulation separately and examinations of word articulation with a picture-vocabulary-test. The test included 46 simple nouns consisting of the most common sounds of the Finnish language, and their combinations in vowels and consonants.

Language behavior was assessed during the interview. Motor speech abilities were controlled by observing various movements of lips and tongue. The tonus and the coordination of the movements were assessed. The rhythmic coordination of hand and eye was assessed by pattern drawing, the appearance of tremor in the lines drawn and unrhythmical movement were observed. Observations were made of disorders in speech rhythm, voice and nasality. Concentration during the test was assessed. The twelve subtests of the psycholinguistic test ITPA were used, but divided between two test sessions so that the auditive subtests were performed by speech therapists and the visual subtests by psychologists.

Cross-disciplinary findings were discussed in a joint meeting, results were compared and the need for special examinations or rehabilitation was estimated and the appropriate measures were taken. Scoring in the logopedic screening tests has been described in detail in the papers of IALP Congress in Washington (Parre, 1980).

For this paper only two items of the total logopedical study are scrutinized and compared with the findings of neurodevelopmental investigations of Bax. Two of the tests will be taken under consideration; namely those which give results of logopedic-linguistic performance of the examined children (Table 2).

1. Articulation (phonological) level

   - sound errors were analysed as errors of phonetic and phonemic features; consonant errors were categorized into distortions, substitutions, omissions, and/or reversals; the vowel errors were counted as substitutions and inversions of the elements in diphthongs.

For assessing the phonological level of the children's speech the analysis was based on the differences of phonetic or phonemic errors in consonants and vowels, their combinations and number of errors per child. The grades of severity were built up of the cumulative occurrence of errors, so that every clinically specific error got one point. (Table 3.)

Table 3. The grades and diagnostic level of articulation.

0 = faultless performance
1 = mild disorder
2 = moderate disorder
3 = severe disorder
4 = very severe disorder

The scale used for articulation skills ranged from 0 to 4.

2. Psycholinguistic level

- psycholinguistic performance was measured by ITPA's auditive subtests and according to ITPA's norms.

3. Neurodevelopmental level

The classification of Bax (Table 4) is divided in this study in three different categories; B1 = 12 (no neurological symptoms), B2 = 13 - 23 (slight to moderate symptoms), B3 = 24 (moderate to severe symptoms) (Saarnivaara & al., 1976). The mean Bax score for the control group was 9.0 (7.5 for girls and 10.2 for boys). The mean neurodevelopmental Bax scores for the risk group were 19.7 (17.7 for girls and 21.3 for boys) (Michelsson & al., 1981).

Table 4. Bax classification

Bax 1 = 12 scores
Bax 2 = 13 - 23 scores
Bax 3 = 24 scores

B1 - B2, Chi^2 29.14, df = 5, $p < .005$
B2 - B3, Chi^2 42.44, df = 5, $p \leq .005$

4. RESULTS

The results have been gathered from the articulation test and psycholinguistic test and are compared with the results obtained from the neurodevelopmental screening test Bax.

As an analysis we present (in Tables 5 and 6) the comparisons and correlations between the findings of the tests of neurodevelopmental level, articulation skill and psycholinguistic abilities.

Table 5. Correlations between BAX scores and articulation.

Grades	Articulation level	Risk children B1 %	B2 %	B3 %	total %	Control group B1 %
0	no errors	73	46	23	48	74
1	one phonetic error	21	33	25	27	20
2	errors in two phonemes (consonants)	3	15	19	12	4
3	errors in different phonemes (vowels and consonants)	2	3	14	6	2
4	many errors in different phonemes, their combinations (at the level of syllable and word structure)	1	3	19	7	
		(140)	(159)	(131)	(430)	(115)

In Table 5 we can see the results of the correlation in the groups B1, B2 and B3 when articulation ability and neurodevelopmental delay are compared. The results show that the more neurological findings there are in children's performance the more disorders they have in articulation and phonological development. Differences

between the performances of the whole group of risk children and the control children are also statistically significant ($p \leq .001$). The scores of group B3 (Table 5) also reveal that the children in this group have a great deal more disorders of grades 3 and 4 than the other children.

A very interesting point in results is the interrelation between the psycholinguistic profile and articulation disorder (grades 3 and 4) among the children who have low neurodevelopmental level B3, and articulation severity is diagnosed in the grades 3 and 4 (Table 6).

Table 6. Psycholinguistic profile and articulation disorders among risk children with neurodevelopmental level (B3)

ITPA Auditive subtests	B 3 No articulation disorders	B 3 Severe articulation disorders
M	34,5	27,9
D	(4,7)	(5,4)
N	30	25

$t = 5{,}81$, $df = 63$, $p \leq 0.001$

5. DISCUSSION

Our approach to this study has been based on the use of test batteries developed for clinical logopedics and neurodevelopmental screening Bax. The aim has been to prove that it is possible to ascertain the influences of the neonatal high-risk factors as retarding elements in development still at the age of five, as well as to clarify that behind slow neurodevelopmental maturity there can be found clusters of severe phonological disorders. The presence of these disorders also correlates with the slow development of the linguistic performance. Two diagnostically very interesting groups appear in Table 6 among the children whose general neurodevelopmental maturation is extremely slow, B3 (N = 55). One group (N = 25) consists of children who have severe articulation problems (grades 3 and 4) and a low linguistic level according to ITPA. The other group (N = 30) consists of children who have no

phonological disorders and their performance level according to ITPA is normal. It means that, as a whole, their syntactic and semantic capability is normal, but the neurodevelopmental maturity of the children is low.

In both cases there are factors based on neurodevelopmental delay; one group consists of children who have developmental language disorder and the other of children with certain psychomotorical delay. Among the control children there does not exist a group corresponding to the one in Table 6. The difficulties in the development of the children are inevitably symptoms of neonatal risk-factors.

It is not easy to define specifically what kind of neurodevelopmental syndromes or language/linguistic disorders exist in each case. Within each speciality and discipline there are many classifications to solve the problem of speech and language behaviour. Many studies have been made to classify developmental language disorders according to linguistic, neurological, psychological, logopedic etc. criteria (Crystal 1980, Ingram 1972, Nettelbladt 1983) and, yet according to Michelsson (1981), the extent to which neonatal disturbances have an influence later in life is not fully known.

Our objective has been firstly to study certain speech and language problems among high-risk children with the screening methods of clinical logopedics and compare the results with the findings of neuropediatrics; secondly to prove that the tests used in screening are valid for cross-disciplinary diagnostics when solving the specific problems and diagnostics in the cases of each children. We have in a way come close to a methodological approach in severe dysphonology and have also applied part of the theory in accordance with Nettelbladt (1983). Although our methods are based more on the clinical logopedics than clinical linguistics we have also found out, as did Nettelbladt (1983), that the focus is too limited for proper understanding of the special features in severe language and articulation problems.

Acknowledgements

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APHASIA SYNDROMES AND THEIR RECOVERY

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1. INTRODUCTION

Throughout the history of the study of aphasia, some sort of system for classifying patients has been used. It is useful initially to characterize different approaches and ask whether or not a syndrome-classification system is still useful in research on aphasia.

An important dichotomy in thinking about classification can be characterized by the terms medical vs. psychometric. The medical view of syndromes is that they are characterized by combinations of symptoms (signs) that are either present or not present. The patient is either anomic or not anomic, apraxic or not apraxic etc.

The psychometric view sees performances as varying along a continuum. Combinations of performance measures gives a performance profile. Arbitrary cut-off points may be used to define syndromes, but statistical techniques for detecting groupings of scores are needed to justify a classification system. The dimensions used for classification are more basic that the syndromes themselves.

The medical and psychometric views also entail a difference in whether syndromes are seen as "real" entities or as convenient fictions. In the medical view a syndrome should be indicative of an underlying pathognomic state or process.

A viable research strategy in aphasiology until recently has been to ask what linguistic deficit is shared by all patients with a clinically defined type of aphasia. There is a recent debate pointing out that all the defining features of a syndrome are rarely present. There is thus only a "family resemblance" between different members of a syndrome. Asking the question "what is the mechanism producing the deficit in Broca aphasia, is it prosodic or syntactic" may thus be meaningless since it presupposes a real core deficit in Broca aphasia which does not in fact exist. Some neurolinguists (Schwartz 1984) have drawn the conclusion that syndromes are therefore uninteresting objects for research.

Poeck (1983) has argued that although syndromes are fictions they are useful fictions, also for neurolinguistic research. Other neurologists, like Kertesz (1979) seem to agree that so long as a classification system allows you to make predictions

about lesion location or prognosis, it is useful in itself. These views reflect a psychometric view of syndromes.

The procedure for identifying syndromes also reflects the conflicting views. Some authors take for granted that there are "real" cases of Broca aphasia, Wernicke aphasia etc., and the trouble is to place a given patient on the map. This may be done intuitively or by a statistical procedure. On the other hand the approach of Kertesz (1979) is taxonomic, which means that he is willing to define a system with sharp boundaries and specific classification rules. The problem "I think this patient is a Broca aphasic, but is he really?" has meaning in the first context, but not in the second because Broca aphasia is fully defined by the classification rules. In the second case it is not the correctness, but the fruitfulness of the classification which is at issue.

In the current literature there thus seems to be a division between neurolinguistically oriented researchers who now want to abandon the classical aphasia syndromes as theoretically uninteresting and clinically oriented researchers, who point out their pragmatic value. The disagreements on classification reflect in my view to a large extent a contrast between a medical and a psychometric view of syndromes. My own view is that syndromes are hypothetical entities which must be defined psychometrically. The fruitfulness of a proposed set of entities and classification rules for research and clinical work must be validated against specified alternatives on a competitive basis. In the further discussion I address two more general questions:

1. Which syndromes should be regarded as valid and how many are they?
2. Assuming that syndromes are clinically useful fictions, are they also theoretically interesting?

2. BACKGROUND

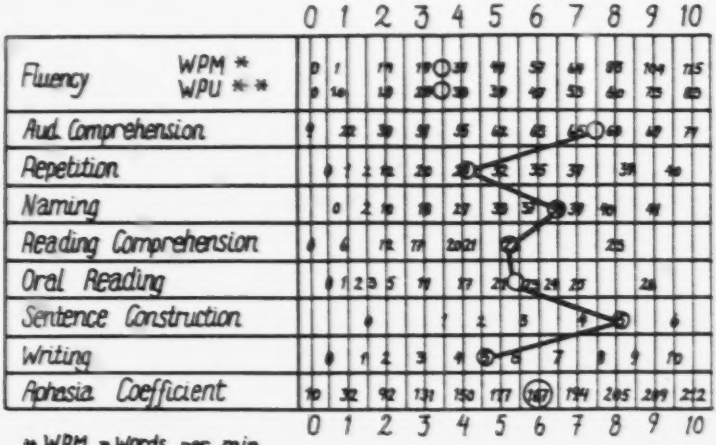
The Institute for Aphasia and Stroke was created by the National Association for Public Health (a private fund) in 1973. The institute was taken over by Sunnaas Rehabilitation Hospital in 1977, but with continued support from the National Association. A program for testing patients was developed and material has been continually registered over a period of about 7 years.

Subjects: 249 aphasic patients have been tested over a period of time between 1977 and 1982. About 130 patients have been followed with repeated testing. The patients are predominantly vascular cases. As far as possible all cases admitted to the hospital with aphasia were tested.

3. CLASSIFICATION

Aphasics are tested with Norsk Grunntest for Afasi developed and standardized in our clinic (Reinvang and Engvik, 1980; Reinvang, 1985). The test includes rating of spontaneous speech, tests of auditory comprehension, repetition, naming, reading comprehension, reading aloud, sentence construction, and writing. The results are transferred to a percentile scale, based on the distribution of scores in a normative group of 161 patients.

Based on statements in the literature and criteria suggested in other tests, definitions with exact quantitative criteria are given for all the classical syndromes in the Wernicke-Lichtheim model. The Wernicke-Lichtheim system of classification has 8 major syndromes and several rare ones. It has been adopted by most research



* WPM = Words per min.
** WPU = Words per utterance

Figure 1. Aphasia test profile in a Broca aphasic.

centers on aphasia (Kertesz, 1979; Goodglass and Kaplan, 1972). In the present adaptation of the system Broca aphasia, for example, is defined as non-fluent speech with auditory comprehension and naming more than 20 percentile points higher than fluency. The following test profile satisfies the criteria for a case of Broca aphasia.

The system is not exhaustive, and unclassifiable cases occur. The view implicit in this procedure is that aphasia types are hypothetical constructs, and that any proposed classification must be evaluated. An attempt was made by my colleagues Sundet and Engvik (1985) to use a statistical procedure (clustering analysis) to evaluate the proposed classification system.

Included in the study were 193 aphasic stroke patients seen at median time since onset of 216 days (range 40 days-7,5 years). The sex ratio was 122 male to 71 female and mean age was 56.5 years (range 22-80 years). Input variables selected for cluster analysis were Fluency, Comprehension, Repetition, Naming and Communication Capacity. Program 2M in BMDP was used with the Euclidian distance measure and the centroid linkage algorithm for joining clusters as options. A discriminant analysis with the same input variables was used to check the validity of the clusters.

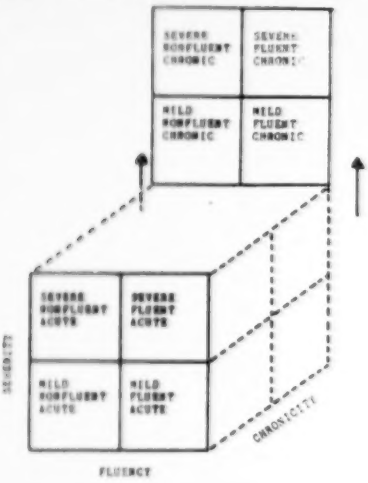
The procedure yields nine distinct clusters and the discriminant analysis shows that two variables can be used to predict the cluster classification with a success rate of 85 %. The first variable combines the measures of Communication Capacity, Comprehension, Repetition, and Naming, whereas Fluency alone formed the second variable.

This analysis suggested to Reinvang (1985) that aphasia types can be reduced to four major categories with a range of severity within each category. This view is in accordance with that of the Aachen group who conceive of four major aphasic syndromes classifying about 80 % of aphasic stroke patients (Poeck, 1983).

For further analysis Reinvang (1985) used the following system (Table 1). The entries in each cell can be conceived as types of aphasia, whereas the marginal terms are the dimensions defining the types.

A traditional argument for using the more detailed Wernicke-Lichtheim system is its alledged ability to predict lesion localization with significant success (Kertesz, Harlock and Coates, 1979). Comparing it to the less detailed classification system just described, I have tested the ability of the two systems to predict lesion localization, and found them to be quite similar. In both cases the relation of function to localization is a probabilistic one. The clinico-pathological correlations give no strong indications for accepting the more differentiated classification system. The analyses are reported in Reinvang (1985, chapter 7).

Table 1. System for grouping of aphasics.



4. RECOVERY

Besides the classically accepted statement that aphasia type predicts lesion localization, what else are syndromes useful for?

Statements about prognosis in aphasia are notoriously difficult to evaluate. If you mean the prognosis of full recovery, then it is surely poor (Reinvang, 1984). If you mean predicting the end state, then surely the most significant factor is the severity of aphasia initially. Severe aphasics are likely to remain more severe than mild aphasics. The statement that prognosis is better in Broca aphasia than in global aphasia is relatively trivial if it means only that relative rank order of severity is usually preserved in recovery.

Is anything known about the magnitude of the recovery, i.e. the difference between the initial and final state? This question raises problems of methodology because difference scores are notoriously unreliable. I note, however, that aphasia test scores show very high coefficients of reliability as measured with coefficients of internal consistency, so that I do not think the results reported here can be dismissed on purely statistical grounds (see also Willmes, 1985).

Using the cube model of Table 1, I have asked which dimensions of the classification predict magnitude of improvement (test-retest difference in the measure of degree of aphasia, the AC). Two time periods are considered, the acute (< 6 months) and the chronic (> 6 months). Analysis of variance is a statistical method that can be used to evaluate this question and it yields the following results in 134 patients (Table 2).

Table 2. Recovery as a function of subgroup.

Group variable	F	p
Type (nonfluent vs. fluent)	1.43	n.s.
Severity (low performance vs. high)	14.27	.001
Chronicity (acute vs. chronic)	5.71	.02

Adapted from Reinvang (1985), p. 141.

All the dimensions of the classification except fluency are the source of significant main effects. Acute patients improve more than chronic and severe patients improve more than mild, when unadjusted difference scores are analyzed. The fact that Kertesz (1979) comes to different conclusions can be explained by his use of recovery scores expressing a ratio of absolute change to maximum possible change.

Analysis of variance has the possibility of analysing for interaction effects. This is the interesting point in regard to syndromes, because a syndrome (like mild, fluent and chronic) in this context is an interaction, or a special combination of values, of the basic dimensions.

Four two way interactions were found, indicating that distinctive subgroups behaved differently compared with the general recovery trend. Some interesting patterns can be pointed out.

The fluents make a much better recovery when the first test is in the acute than in the chronic stage, whereas for the non-fluents this is only a slight tendency. Recovery is thus less dependent on time after injury in non-fluents, as has also been found by Sarno and Levita (1979).

The severely aphasic patients make a high recovery score when the first test is in the acute stage and a poor score in the chronic stage. The mild aphasics make about the same recovery regardless of time after lesion. Recovery is thus less dependent on time after injury in mildly aphasic patients.

5. PROGNOSTIC SIGNIFICANCE OF NEUROPSYCHOLOGICAL FINDINGS

To further evaluate the hypothesis of distinctive recovery patterns, information about results on other neuropsychological tests is introduced.

All aphasics are tested with a standard neuropsychological battery. It is a mixture of standard psychometric tasks, tests from well known neuropsychological batteries and tests from the experimental neuropsyhological literature (Table 3.)

Table 3. Overview of neuropsychological tests.

Function	Problem area	Test
	Verbal immediate memory	Digit span Pointing span
LANGUAGE	Verbal learning	Suprespan learning
	Verbal conceptual function	WAIS-Verbal IQ Paired associate learning
	Ideational apraxia	Object use
MOTOR FUNCTION (APRAXIA)	Ideomotor apraxia	Movement imitation
	Motor coordination	Grooved pegboard Finger tapping
	Visual immediate memory	Corsi blocks
VISUAL ABILITIES	Visual route learning	Suprespan Corsi blocks
	Problem solving	Raven CPM WAIS-Performance IQ
	Construction	Copying

On the basis of factor analytic studies (Reinvang & Sundet, 1985) my present view is that aphasia is a multi-dimensional neuropsychological deficit in which language, motor performance and visual abilities are identifiable dimensions. Are the non-verbal dimensions of aphasia prognostically significant with respect to recovery of language? Since recovery varies in subgroups, the analysis follows the classification system already proposed.

A summary of the results of these analyses is given (Table 4). The fluent versus nonfluent distinction is omitted because this variable showed no main effect on recovery. The groups defined by the significant interaction terms were not analyzed because the number of subjects in each group was small.

Table 4. Prognostic neuropsychological signs.

Subgroup	Predictive function or problem area
SEVERE	Motor function (ideational apraxia, ideomotor apraxia, and coordination))
	Verbal immediate memory
	Visual immediate memory
MILD	Construction
	Ideational apraxia Ideomotor apraxia
	Visual route learning
ACUTE	Verbal immediate memory
	Ideomotor apraxia
CHRONIC	Ideomotor apraxia Coordination

Adapted from Reinvang (1985), p. 143.

Correlations derived by such an explorative procedure should be interpreted with reserve. If we bear in mind the factor analyses of neuropsychological tests, it is reasonable to require that several tests representative of a factor show significant correlations before this factor is taken to have a significant prognostic value.

Using the factor analyses as guidelines for interpretation, one may conclude that apraxia was a prognostic sign in both severe and mild aphasics. The prognostic relation had the opposite sign in the 2 groups, and therefore the effects cancelled each other out in an overall analysis.

Verbal immediate memory (represented by two tests) was also a prognostic sign in severe aphasia but not in mild aphasia.

The visual non-verbal tests do not appear to be strongly correlated with prognosis although scattered significant correlations are found.

The clearest prognostic implications of neuropsychological functions appeared in relation to the severe vs. mild distinction. The findings are not so readily interpretable with respect to the acute versus the chronic distinction. Low but significant correlations on a scattered selection of tests are difficult to assess, but the general point of subgroups is supported.

6. RELATIONS BETWEEN FUNCTIONS AT DIFFERENT STAGES OF RECOVERY

Is the functional domain measured by the different tests differently organized in acute and chronic patients? The question is not about the content, but about the structure of the preserved functions. To answer it a factor analysis of the aphasia test results and the neuropsychological tests is performed. Consider a hypothetical contrast between two possible modes of functional organization.

Case 1: The brain is a single channel of processing, and all tasks are performed by the same neurological and functional devices.

Case 2: The brain has a number of well differentiated processing mechanisms that can perform different functions.

If studied with factor analysis case 1 should be functionally characterized by a single factor accounting for all the variance in performance. Case 2 should show several functional factors and absence of a prominent G (or general) factor.

When applied to the severe or mild, and chronic or acute patients, the pattern observed is that severe and acute patients show a smaller number of different factors and a higher G-factor, whereas the mild and chronic show a higher number

of different factors and a smaller G-factor (see Reinvang 1985, chapter 8). It was thus the severely aphasic group that showed the clearest trend in the direction of greater differentiation of function with time.

Summarizing the results on recovery I found that the severe aphasics made a greater-than-average recovery, especially if, in addition to being severe, they were in the acute stage and young. Several neuropsychological variables representing factors of apraxia, verbal memory, and nonverbal memory were predictive of recovery. Finally, the severe aphasics showed signs that a process of functional differentiation was taking place between the acute and the chronic stages of the illness.

The finding of distinctive patterns over time and across functions may indicate different recovery mechanisms in different subgroups. Because the differences between groups were not dramatic, it is more reasonable to conclude that several recovery mechanisms may have operated simultaneously, but with different weights in different groups.

7. DISCUSSION

What theoretical conclusions (or speculations) can be advanced? In his influential review and critique of current classification approaches, Caramazza (1984) considers problems for the view which he himself advocates, namely that well motivated theories of normal performance should be the basis for analyzing pathological phenomena. The **fractionation** assumption is "the belief that brain damage can result in the selective impairment of compontents of cognitive processing" (p. 10). He says that "a less than optimistic view of the interpretation of pathological performance is that even if the fractionation assumption were to be true, the remaining, unimpaired processes will work differently when one component is not functioning normally. In this case the pathological performance would not have a transparent relation to the working of the normal system, and would make the analysis of pathological cases irrelevant for the understanding of normal cognition" (p. 11). He then goes on to dismiss this possibility by an act of faith, since it is threatening to his whole program.

I think the cognitive-linguistic approach to neuropsychology is in general fruithful, but not sufficient as a theoretical model. Let us ask if Caramazza's problem does not admit of a solution which does not invalidate his approach.

Some now almost forgotten classical papers on aphasia focus on the qualitative changes in cerebral function taking place after injury. I have in mind the work of Goldstein and his concept of loss of differentiation (dedifferentiation) after injury (Goldstein, 1948). The resulting effect on behavior is concreteness, rigidity, and stimulus bound responding. Although he goes too far in generalizing this principle, I think it should be brought back into our theories of aphasia. In a large cerebral lesion producing a severe degree of aphasia, my results can be taken to show that a closer linkage (loss of differentiation) exists between language and non-verbal neuropsychological functions. Other clinical signs of this loss of differentiation are the context-dependency of the patient's responses and his inability to process two things at the same time.

Some functional interpretations may be suggested for the three dimensions I have used to define the aphasic condition.

The fluency dimension is related to a specific kind of information processing mechanism. There seems to exist a localized and functionally distinct mechanism for maintaining normal fluency of speech. Injury to this mechanism produces nonfluency. Except for fluency I don't think we have any strong indication that other aphasic phenomena reflect processing mechanism in any transparent way. I specifically do not think that auditory comprehension, as measured in tests of aphasia, reflects any well defined linguistic processing mechanism with a neurological correlate.

The severity dimension must of course reflect failure of specific processing mechanisms, but also the loss-of-differentiation aspect in which the brain becomes more like a single channel device. The multi-dimensional nature of severe aphasia partly reflects the undifferentiated response of preserved brain areas to varying functional demands.

Chronicity is linked with the kind of recovery mechanism which is operating. One class of such mechanisms is the relearning or reorganization processes. These are similar in that both presuppose a stable underlying processing system, but one focuses on strategies whereas the other does not try to analyze the system. This class of mechanisms is operative in chronic aphasia.

The other class of processes, which are more active in acute aphasia, contains relocalization and what I will call re-differentiation of function. This supposes that some degree of readjustment of the function-to-structure relationship takes place. The idea that one area of the brain takes over the function of another is, I think, biologically unmotivated. My final exercise will be to show an example of how re-

differentiation can operate to establish a partly new pattern of cerebral organization without involving ideas of arbitrary shuffling around of functions.

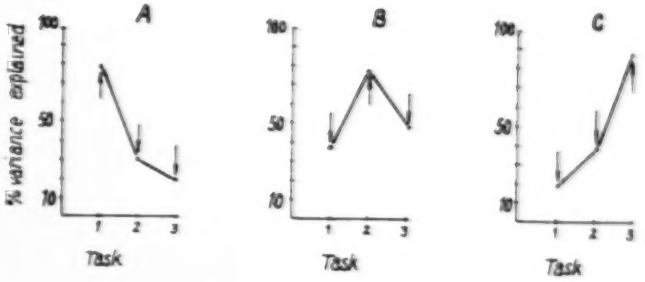


Figure 2. Differentiation of function in three areas (for explanation, see text).

Figure 2 represents a hypothetical system consisting of three devices with different, but overlapping functional profiles. The devices can be seen as schematic representations of brain areas. The three devices communicate with each other, and as a result of this interaction the functional profiles are sharpened in the direction that functions which one device performs better than any of the others are boosted, and less efficient functional capacities are supressed. With an injury to one of the devices in such a system the basic functional profile of the uninjured devices would remain unchanged. It is easy to imagine, however, that part of the functional profile of a remaining device might now be boosted rather than suppressed because it is the most efficient device with respect to that function in the residual system. This argument is pursued in greater detail in Reinvang (1985, chapter 9).

These then are the kinds of recovery processes at work in chronic and acute aphasics. If we knew more about how to stimulate them, we might come closer to a rational therapeutic approach. This is especially needed in relation to the severe aphasics, a group which in my clinical judgement does not profit well from therapy based exclusively on an information theoretical componential analysis of their deficit.

I do not share the fear of Caramazza that accepting the sort of model I have suggested will invalidate the componential approach to the study of information processing. We study phenomena along a continuum, and there is no sharp breaking point at which we must throw up our hands in dispair. If we can study cognition in non-human species then it surely cannot be a precondition for the study of aphasics that they can be modeled as normals-minus-one. The analogy intended is to music-minus-one, which is a recording of a concerto without the solo instrument. Without carrying the analogy too far I suggest that in applying it to loss of language after brain injury the resulting concerto becomes rather a different piece with the other instruments having to adjust their functions without loosing the organizing perspective of producing music.

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